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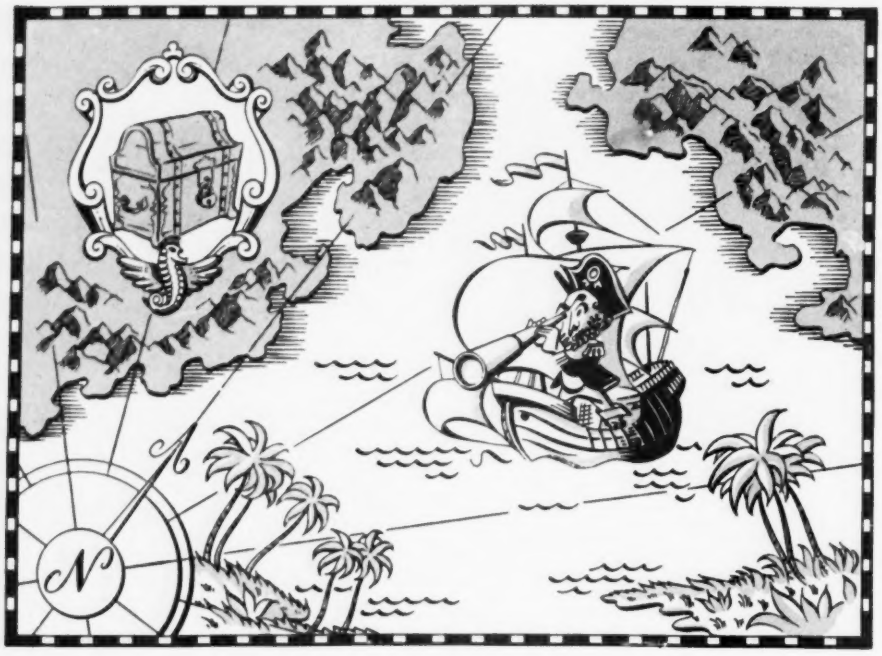

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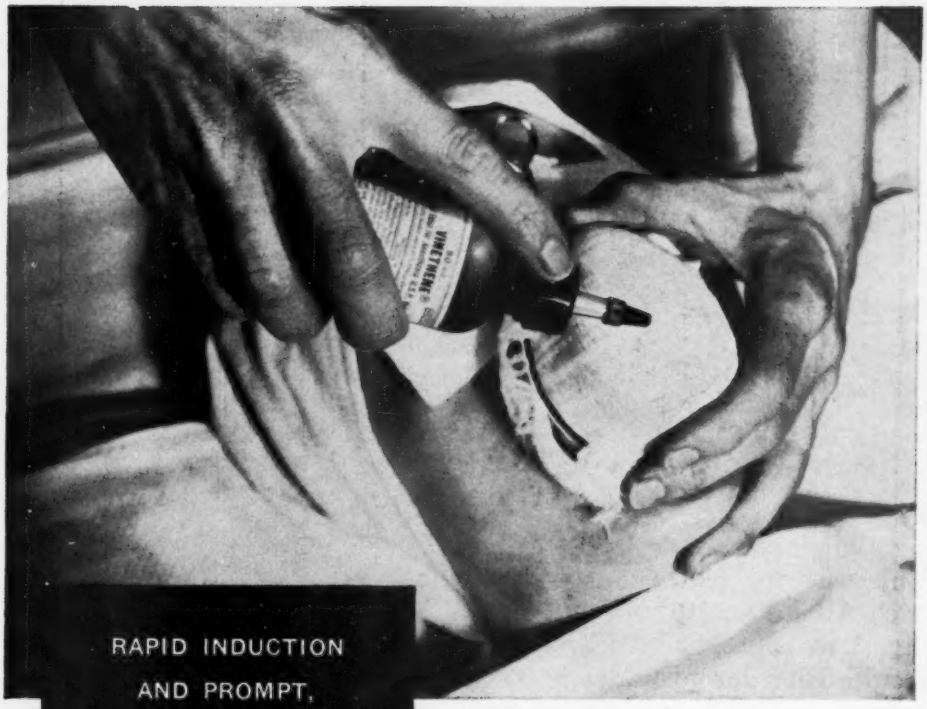

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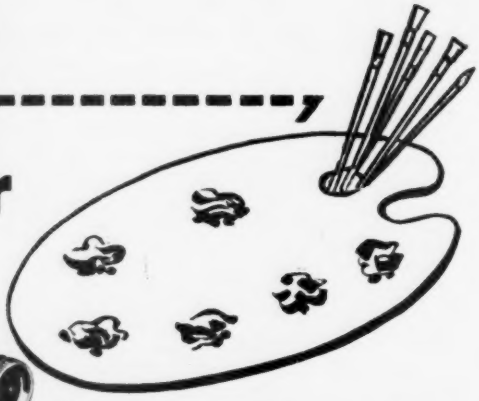

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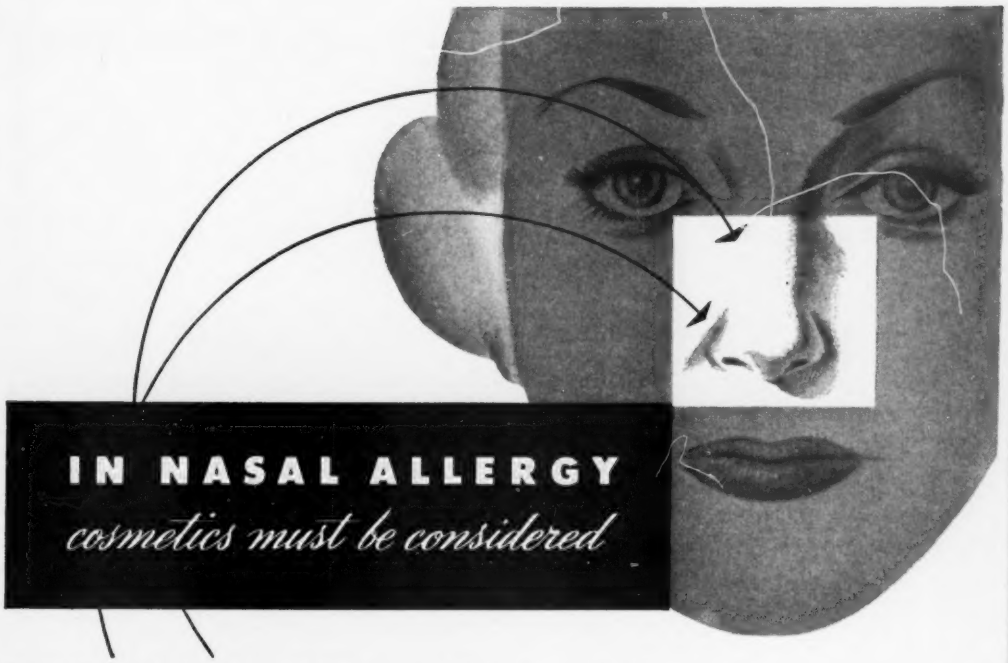

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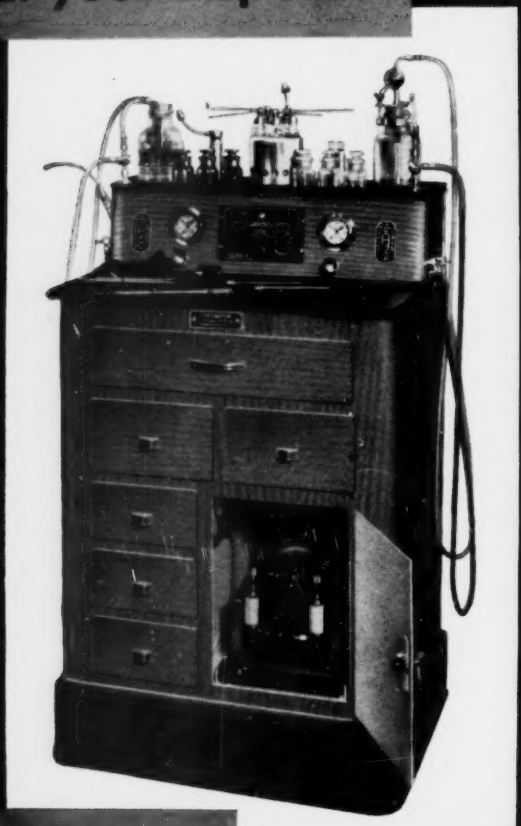

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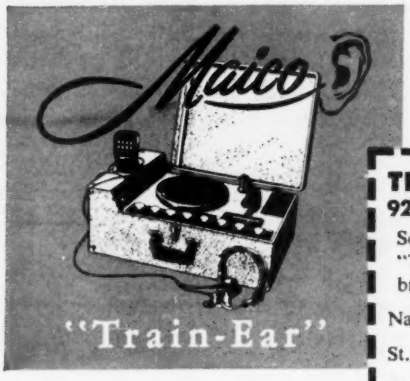

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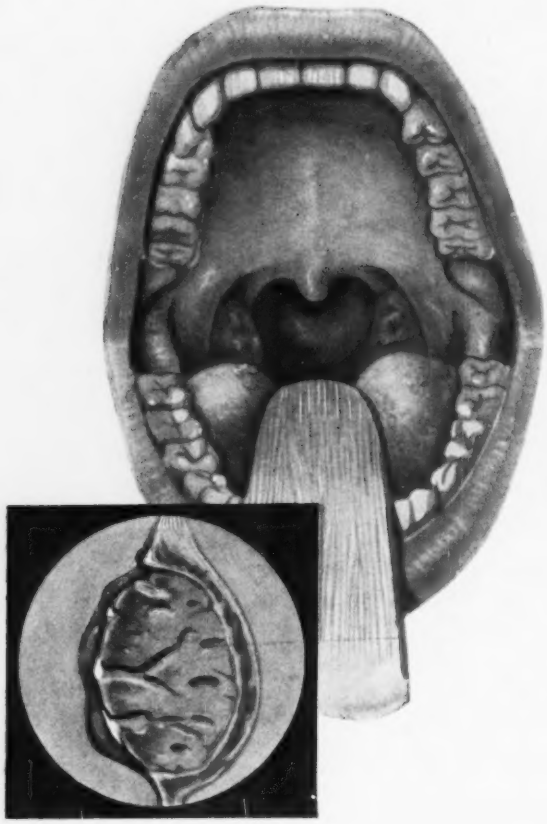

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VOLUME 55 FEBRUARY 1952 NUMBER 2

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DIAGNOSIS OF DEAFNESS IN THE VERY YOUNG CHILD

FREDERICK R. GUILFORD, M.D.
AND
C. OLAF HAUG, M.A.
HOUSTON, TEXAS

THE DIFFERENTIAL diagnosis of deafness in the very young child is one of the most challenging and formidable problems facing the physician today. The age group of the very young child, for the purpose of this paper, falls within the range of 2 to 6 years. A complete prenatal and postnatal history, an otorhinolaryngological examination, an evaluation of the total behavioral symptomatology, and batteries of tests must be employed in order to assess the hearing function adequately or affix the cause of the communication disorder.

The ultimate success of early language and speech development is dependent on the commencement of appropriate early training procedures following the accurate diagnosis of the impairment. It is generally recognized by educators that, in the absence of accurate diagnosis of the impairment, either therapy will be delayed or the wrong type of therapy may be applied. In the usual clinical situation much valuable time is lost before therapy is initiated, owing to failure to obtain accurate and reliable audiograms and to the prevalent belief that these audiograms are unobtainable at this early age. Often when the physician is confronted with this situation, the parents of the child are told to return for further evaluation when the child has reached the age of 5 or 6 years, at which time a reliable test can be made. This is indeed unfortunate, inasmuch as the hard-of-hearing child's entire language and academic and social development hinge on the opportunity of getting early training, preferably before 2½ years of age. With the conventional audiometric technique it is impossible to obtain an accurate evaluation of the hearing in the very young child, since the listening situation, as usually carried on, is a meaningless experience to the child. In addition, the child does not fully comprehend what is expected of him in the way of responses, and the short attention span at this age makes it difficult or impossible for the child to attend for an adequate length of time for good audiometric test results.

The problem is further complicated by the fact that lack of response to auditory stimulation does not necessarily indicate a hearing impairment. It must be recognized that several other conditions may give the same lack of response to auditory stimuli. The conditions oftenest confused with deafness are mental retardation, "aphasia," and emotional disorders. The proper evaluation of hearing impairment is one of the initial steps in the differential diagnosis of these conditions. The

Read before the Section on Laryngology, Otology and Rhinology at the One Hundredth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1951.

From the Hearing Clinic, Department of Otolaryngology (Dr. Guilford), and from the Department of Audiology and Speech (Mr. Haug), University of Texas School of Medicine, Galveston, Texas.

remainder of the process in the differential diagnosis of these conditions involves further tests, the description of which is beyond the scope of this article.

A number of attempts to overcome the serious shortcomings in conventional audiometric technique when applied to children of this age group have been made. While many of the methods attempted are extremely ingenious, we feel that serious shortcomings exist for their practical use in the clinical situation because of the prohibitive expense, complexity of interpretation, length of time required for the tests, possibility of confusion of test instructions by the child, and lack of dramatic and attention-holding appeal for the child.

1. The Ewing [1] electric train-tunnel test situation is cumbersome to handle, lengthy in administration, and lacking in plausibility of sound source and sound stimuli.

2. The Bloomer [2] picture-card test is lacking in attention appeal and affords the possibility of confusion in directions.

3. The Dix and Hallpike [3] peep-show test again is lacking in dramatic, attention-holding appeal and does not afford selection of unilateral impairments as it is presented in the free field. In addition, this test requires elaborate and space-consuming equipment.

4. The Marcus and Gibbs [4] electroencephalography technique is costly and requires highly skilled technicians and special test surroundings. It is, in addition, very complex in its interpretation and requires extensive analysis of results.

5. The psychogalvanometric technique, as described by Bordley and Hardy,[5] is extremely costly, requires special test surroundings, and must be operated by highly skilled technicians. Further criticisms which have been made are that below 50 db. poor responses are obtained, that frequencies above 2048 and below 128 cycles give feeble galvanic skin reflexes, and that inconsistency of response is frequent.

6. The noise-maker tests described by Utley,[6] Ewing,[7] and Mykelbust [8] are qualitative and not quantitative to any degree and are difficult to evaluate with any degree of accuracy. In addition, the tests are given in the free field and do not afford unilateral evaluation.

An attempt has been made to alleviate many criticisms of the above tests through the use of an accessory unit to motivate and interest the child during the pure-tone situation. This device, developed by one of us (C. O. H.), is called the Pediacoumeter. It is readily portable, is not expensive to produce, has great attention-

1. Ewing, A. W. G.: Aphasia in Children, New York, Oxford University Press, 1930.

2. Bloomer, H.: A Simple Method for Testing the Hearing of Small Children, Quart. J. Speech **28**:343 (Oct.) 1942.

3. Dix, M. R., and Hallpike, C. S.: The Peep-Show: A New Technique for Pure-Tone Audiometry in Young Children, Brit. M. J. **2**:719 (Nov. 8) 1947.

4. Marcus, R. E.; Gibbs, E. L., and Gibbs, F. A.: Electroencephalography in the Diagnosis of Hearing Loss in the Very Young Child, Dis. Nerv. System **10**:170 (June) 1949.

5. Bordley, J. E., and Hardy, W. G.: A Study in Objective Audiometry with the Use of a Psychogalvanometric Response, Ann. Otol. Rhin. & Laryng. **58**:751 (Sept.) 1949.

6. Utley, J.: Procedures for Examining Very Young Acoustically Handicapped Children, Chicago National Society for Crippled Children and Adults, 1949.

7. Ewing, I. R.: Deafness in Infancy and Early Childhood, J. Laryng. & Otol. **58**:137 (April) 1943.

8. Mykelbust, H. R.: The Differential Diagnosis of Deafness in Young Children, J. Exceptional Child **17**:97-117 (Jan.) 1951.

holding appeal to the child, is easily administered in the clinical situation, and does not require special test conditions for its administration. In addition, the test results can be easily interpreted by all clinicians.

THE PEDIACOUMETER AND ITS USE[9]

The Pediacoumeter is an electronically powered, solenoid-operated, portable device to be used in conjunction with the conventional audiometer for pure-tone testing of children. It is an attempt to overcome the serious disadvantages of other instruments and tests in use today—disadvantages of high cost, excessive time and complexity of administration, difficulty in understanding directions, lack of dramatic and interest appeal, narrowness of hearing range tested, complexity in interpreting results, lack of individual ear evaluation, and lack of correlation between the test sounds presented and the objects represented as the sound source. It is an attempt to make a practical, easily administered, reliable clinical tool for audiometry with children.

Seven jack-in-the-box heads have been appropriately modified to represent each of the audiometric test tones presented, and attached to solenoids for release by the push-button switch in the hands of the child testee. In addition, a solenoid switch is provided for the tester. These additional switches in the line, one in each of the solenoid circuits, provide a means of checking and controlling the indiscriminate, random completion of the circuit by pushing the button to gain a favorable response. With this arrangement it is necessary to have both of the switches closed in order to operate the solenoid and release the jack-in-the-box.

There are two main tests used with the Pediacoumeter: the normal test and the conditioning test.

The normal test is used when the child has sufficient maturity (the usual age is over 3 years) and when there are no indications of total hearing loss or mental disturbance. In this test the child is told that he or she is to play a game with the operator. The instructions run something like this: "Now, 'Lana,' we are going to play a game with these little houses. In these houses there are some little people all locked up. They can't get out unless you help them. Now when they holler to get out, I want you to let them out by pressing this button [placing push button in the child's hand]. Sometimes they're sleeping and are very quiet—then they don't want to get out. If you press the button then, nothing will happen. But just as soon as you first hear them holler to get out, you press this button, and then the little people will come outside. Now I'm going to put these phones on you [placing headset over testee's ears], so you'll be sure to hear these people when they cry to get out."

The operator then sweeps up quickly until he finds the general area or range within which he must work. Then he begins to probe with 5-db. decrements in the intensity of the tonal stimulation, presented in short bursts of sound from silence. Only when the tone is actually being presented to the child is the appropriate switch on the switch box closed, thus making possible the release of a given jack-in-the-box. Whenever the tone is not presented, the switch on the switch box is immediately opened, so that if the child then presses the push button only the red light will go on. This action is then apparent to the operator, and he can caution the child against further playing and remind him of the procedure in the

9. The Pediacoumeter and its uses are described by Mr. Haug.

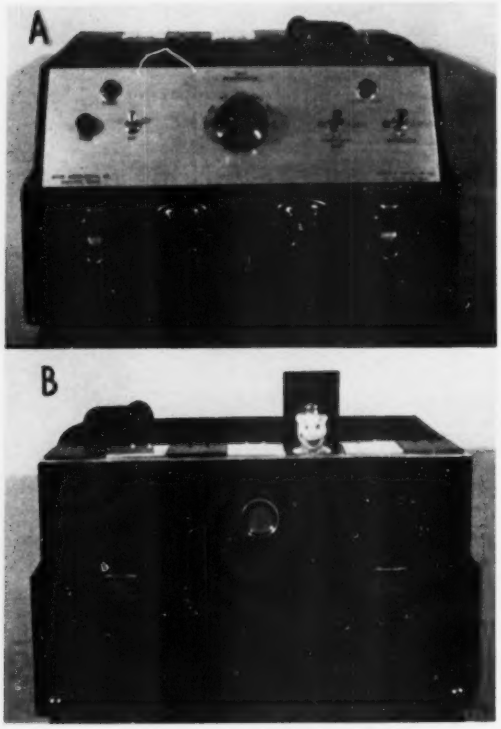

Fig. 1.—*A*, front view of Pediacoumeter; *B*, Pediacoumeter with one jack-in-the-box released.

Fig. 2.—Administration of the Pediacoumeter test with a young child reacting to a little man that she has just released after hearing him "call to get out."

game. After the idea of the game is established, two trials are made at each frequency in one ear, after which the process is repeated in the other ear.

The conditioning test is used when a too low chronological or mental age interferes with the comprehension of instructions for the regular test, when indications point to possible total deafness, or when too low auditory-perceptual maturation levels prevent ordinary response to pure-tone stimulation. Here the intention is not to obtain a definite audiogram of the child's hearing, but merely to determine whether he can hear and generally what the degree of impairment is. With the light switch in the "conditioning" position, a test tone is presented to the subject, well above normal threshold, at the same time closing the switch for the appropriate jack-in-the-box figure. This process is then repeated several times until the subject, by his own operation of the push button, has come to realize that in the presence of stimulation by sound and/or light (at this point it is not known to which he is conditioned), pushing the push button makes for a favorable response, i. e., the release of a jack-in-the-box. The light is then removed, and if the push-button response persists in the presence of the same auditory stimulation, it is safe to assume that the subject is a hearing child. The inverse deduction, that if the subject does not respond after removal of the light, he is therefore a deaf child, cannot be made, however. All that can be said is that he probably has not been properly conditioned to the sound.

The two trials of the regular Pediacoumeter test procedure were compared with two trials of standard audiometric procedure without the Pediacoumeter on 20 subjects between the ages of 2 and 6 years. The order of presentation of the tests was varied to eliminate "practice effect" in the comparison.

For all ages and subjects tested, the Pediacoumeter has had considerable interest appeal; many children repeatedly expressed a desire to "play this game again" and thanked the operator for the fun they had. This dramatic, interest- and attention-holding factor, and the ease with which children "caught on" to what was expected of them, evidenced themselves in a sharp contrast between the consistent and plausible audiometric curves obtained with the Pediacoumeter and the bizarre, inconsistent ones arrived at by using only the audiometer.

The Pediacoumeter threshold difference range on retest was 0 to 10 db. for any given frequency. The average difference range over all the frequencies on retest was 2.1 to 4.2 db. Without the Pediacoumeter, the threshold difference range on retest was 0 to 50 db. The average difference range over all the frequencies on retest was 19.2 to 34.3 db.

In addition, the Pediacoumeter has had great clinical success in obtaining audiograms on children with severely impaired hearing for whom results had heretofore been highly unreliable or unobtainable.

Hermann Professional Bldg.

ABSTRACT OF DISCUSSION

DR. SAM H. SANDERS, Memphis: The Secretary, in planning to create interest in the title of this paper, changed it a little after writing the author and receiving his approval. We should like to explain that. Dr. Guilford has been doing a great deal of work on the diagnosis of deafness in young children. Mr. Haug, in working on his thesis for the degree of Doctor of Philosophy at Galveston, where the University of Texas School of Medicine is located, has developed this instrument. The most unusual thing about it is the way in which he has developed it and the cost. If I am not mistaken, he had this instrument made, after his planning and

arranging it, for a cost of about $125. If you look at it closely, you will wonder how he could fix something that has all the qualities it has at such a small cost. These young men are to be congratulated on the work that they are doing in that section of the country.

DR. STANLEY H. MACHT, Hagerstown, Md.: An otolaryngology section meeting is an unusual spot for a radiologist to discuss a paper. I offer my comments neither as a radiologist nor, for that matter, as a physician, but as the father of a child for whom the diagnosis of a marked hearing loss was made by one of the psychogalvanometric skin reflex tests.

When my son was 2 years old, I was informed that he had an 80-db. hearing loss in one ear and a 60-db. loss in the other. We were supposed to prepare him for lip reading and institutional care. At the age of 3 the child has no hearing loss. He now talks, is fine, and has no abnormalities other than a minor behavior problem. This, of course, may reflect upon his parents.

Because of this personal experience, I should like to interject a word of caution that one be extremely careful about making a diagnosis of marked hearing loss in the very young child. Even the "reflex" tests may be wrong. The parent whose child has had such a diagnosis undergoes a terrific psychic trauma.

DR. FREDERICK R. GUILFORD, Houston, Texas: We certainly agree with Dr. Macht concerning his unfortunate experience with his child, and we do think that a diagnosis on a preschool age child should be made with accuracy and caution. We hoped that we had stressed that point.

Concerning this instrument, I am sure that Dr. Fowler's remarks are justified, in that he has a method of making the diagnosis in young children which is similar to the present method, this one being a mechanical device. In the hands of an experienced operator in the hearing-clinic situation, this machine can be used for more than the quantitative analysis of hearing. It also brings out certain features of the problem of differential diagnosis. The child's response to the controlled situation may give information in addition to the amount of hearing loss present.

DR. EDMUND P. FOWLER JR., New York: This new gadget looks as though it might be very useful for those children that cannot be reached by a simpler type of technique.

We have been using in the Presbyterian Hospital in New York for several years movable toys or hand puppets, some of which can be obtained from a well-known breakfast food company for 25 cents. They are called Snap, Crackle, and Pop. One can quickly become sufficiently adept at handling these puppets with one hand and the audiometer with the other. Our technique requires the mother, the child, and the audiometer. The mother faces the child, who usually sits on the floor with his back to the—I was going to say—"puppeteer"; that is probably the word. She tries to keep the child's attention until the fascinating puppet starts to make faces or wiggle its nose. This occurs immediately after a sound has been presented. If the child hears, he usually turns around to see the puppet. After an appreciable interval the puppet disappears behind a piece of cardboard or some other screen, and the mother then takes the child's attention until the "show" is again announced by the presentation of another note.

All of these techniques are similar. I don't think it makes very much difference whether you ask the mother to teach the child to move a block, as recommended by Westlake and others, or to take a block and drop it in a bag, or to take the block out of a bag and throw it on the floor. Any such technique using a reward for the conditioning is very useful and, in our opinion, better than psychogalvanometry. The latter, I think, should be reserved for special cases, because it requires not only expensive apparatus and a trained team but also a cooperative child. Many of these children are not cooperative unless you play games with them, and they are often very uncooperative if you annoy them with an electric shock. This Pediacoumeter can be added to the hearing test armamentarium if you find that it is easier for you to use; but I suggest that you can train your mothers and train yourself to play simpler games with the children. Within the same amount of time you can get a threshold within 20 db.

I, like Drs. Guilford and Haug, have never been able to understand why the psychogalvanometer will not give responses below 500 or above 2,000 cycles with any degree of accuracy. I think the apparatus described by the authors and the block and puppet games are very much simpler. Although all methods tend to take a great deal of time and skill, they are rewarding in the end. One cannot start to educate young children until a hearing threshold is definitely established. Nor can one differentiate aphasia, mental deficiency, and hysterical deafness without conditioning methods.

LOUDNESS PERCEPTION FOR PURE TONES AND FOR SPEECH

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THE USUAL test for loudness perception of pure tones is the audiogram, describing the auditory threshold as a function of frequency. In the case of loudness perception for speech, the usual examination is with the whispered voice or, more recently, the phonograph audiometer. Whatever the type of sound, the examiner attempts to determine the weakest perceptible loudness.

There is, however, another aspect of loudness perception in which threshold data may be uninformative. This is the ability to perceive and judge the loudness of sounds whose intensities are well above threshold. Techniques other than threshold audiometry must be used to explore this aspect of hearing.

It is well recognized that with the normal-hearing person as the physical energy of a sound increases its loudness (as a distinct psychological experience) increases at a different rate. The growth of loudness for the normal ear is now well specified for most pure tones [1] and very tentatively specified for speech.[2] However, as energy increases, the growth of loudness for ears with disturbances in hearing may take a quite different course from that for normal ears. Particularly with the so-called perceptively deaf ear, the loudness of a sound (as its energy is increased over threshold strength) may increase in striking fashion within the space of only a few decibels. The importance of carefully assessing the usefulness of this phenomenon is apparent.

An unusually rapid growth of loudness on the part of certain ears was observed many years ago by Habermann.[3] Working with boiler makers, he found it "worth noting that loud speech was heard relatively much better than whispered speech, for which hearing was greatly diminished." A number of authors have followed Gradenigo [4] in using the index vocalis (ratio of whispered-voice to spoken-voice reception) to differentiate a certain type of abnormality in hearing. The index

From the United States Naval Medical Research Laboratory.

1. Fletcher, H., and Munson, W. A.: Loudness, Its Definition, Measurement, and Calculation, J. Acous. Soc. Am. **5**:82-108, 1933.

2. Pollack, I.: The Effect of White Noise on the Loudness of Speech of Assigned Average Level, J. Acous. Soc. Am. **21**:255-258, 1949.

3. Habermann, J.: Über die Schwerhörigkeit der Kesselschmiede, Arch. Ohrenh. **30**:1-25, 1890.

4. Gradenigo, G.: Studi e proposte di acumetria: Indice vocale (Index Vocalis) auditus, Arch. ital. otol. **23**:177-208, 1912.

vocalis, however, as a consequence of the inherent unreliability of speech-reception testing with the free voice, has had little quantitative significance.

Fowler [5] first turned the resources of the modern audiometer to the question and devised numerous ways to examine the growth of loudness of pure tones at suprathreshold intensities.[6] To the abnormally rapid growth of loudness in certain ears he gave the name "recruitment" [7] and demonstrated its usefulness as a diagnostic sign in perceptive deafness.

Of all the methods of investigating recruitment which Fowler devised, among them being (1) studies of the intensity-discrimination threshold, (2) effect of masking, (3) monaural loudness balancing between two frequencies, and (4) binaural loudness balancing with one frequency, a form of the last was found most reliable and was most extensively used. This method, the only one used in the present experiment, will be described fully in the section on procedure.

The practical significance of recruitment is largely in speech reception. It is obviously important to know whether an ear with a depressed threshold audiogram would overcome this handicap for speech at 50 to 70 db. above threshold. Fowler assumed, after examining some hundreds of patients with pure-tone recruitment, that many ears did overcome this handicap. He concluded that, if one could demonstrate recruitment in a patient or could infer its presence by showing a perceptive component in the hearing loss, the patient should be given additional credit for speech reception over and above what one would predict from the air-conduction audiogram alone.

There are, however, at least three assumptions contained in this line of reasoning, none of the three being fully documented. They may be put as questions susceptible to experimental test. First, granted that recruitment exists for pure tones with some patients, does it exist for other kinds of sound, particularly the extremely complex sounds of speech? Second, is it true that all perceptive-deaf patients show recruitment? Third, if recruitment does occur for speech in the same way as it does for pure tones, does this increased loudness of speech actually improve its intelligibility? Each of these assumptions must be investigated before the clinical importance of recruitment in speech communication is fully understood.

The present paper examines the question whether the growth of loudness for pure tones and the growth of loudness for speech follow similar courses. The other questions (whether recruitment can be expected in all cases of perceptive deafness and whether recruitment improves intelligibility) will be left largely to subsequent papers.

Although the study of recruitment is a rather recent development in audiology, already several theories have been advanced to account for its occurrence. However, data sufficient to begin to discriminate among theories have not yet been accumulated, and another aim of this paper is to provide further data from clinical

5. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. **8**:151-155 (Aug.) 1928.

6. Fowler, E. P.: A Method for the Early Detection of Otosclerosis, Arch. Otolaryng. **24**:731-741 (Dec.) 1936.

7. Fowler, E. P.: The Diagnosis of Diseases of the Neural Mechanism of Hearing by the Aid of Sounds Well Above Threshold, Tr. Am. Otol. Soc. **27**:207-220, 1937.

patients and data from normal-hearing subjects from whom analogues of recruitment may be recovered with the use of such techniques as occlusion, fatigue, and masking.

EXPERIMENTAL STUDY

Apparatus.—The equipment used in this study is shown in Figure 1. The output of a pure-tone oscillator was led to a transformer which divided the source into two separate channels, one channel leading to an attenuator and then to one side of a right-left switch and the other channel leading to two attenuators connected in series and then to the other side of the same switch. The experimenter had full control of both channels by attenuators No. 1 and No. 2; the patient controlled attenuator No. 3 in the channel leading to his worse ear. Two matched Permoflux PDR-8 headphones, which have a flat frequency-response characteristic through the speech range, mounted on a wide-band headset and equipped with supra-aural cushions, were connected to the switch.

The equipment for speech was the same, with a high-quality phonograph turntable and amplifier in place of the oscillator. The records used were part of a news broadcast by Fulton Lewis Jr. and a 20-minute recording from the book "Maneaters of Kumaon," read by one of us (J. D. H.).

The patient sat in a soundproof room. The playback was in a separate room, eliminating the possibility of a patient's hearing the pickup directly.

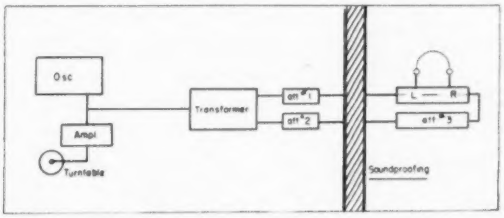

Fig. 1.—Apparatus used in study of loudness perception for pure tones and for speech.

Subjects.—Thirty-one subjects were used in this experiment. Eight were normal in hearing acuity, ± 5 db. for the six octaves 256 to 8192, according to tests with a 6BP Western Electric audiometer. Four of the group of normal subjects were used to calibrate the speech system. The other four were experimental subjects who had one ear plugged by an ear warden to simulate conductive deafness. The remaining 23 were patients who had some degree of monaural deafness, representing a variety of types of hearing defect.

Procedure.—The patient was comfortably seated facing the right-left switch and attenuator No. 3. After he had adjusted his earphones, a threshold was obtained for his better ear. He was then asked to manipulate the attenuator and switch and decide at what setting he barely heard the tone or the voice, as the case might be, in his worse ear. This was repeated five times, and the judgments were averaged. He was told that his attenuator settings from judgment to judgment might well be different, as the experimenter could, unknown to the subject, manipulate another attenuator connected in series to the one the subject was using.

After thresholds were calculated for each ear, the sound was set at 10 db. sensation level in the patient's better ear. He was then asked to match that loudness in his worse ear by moving the switch back and forth at will (so that the sound was heard in the two ears alternately), adjusting the attenuator which controlled the intensity of the sound in his worse ear. Five equality judgments were averaged. The sound in the better ear was then successively set at 20, 30, 45, 60, and 75 db. sensation level; five equality judgments were made and averaged at each level. Finally, differences in sensation level between the two ears at points of equal psychological loudness were graphed. From the course taken by these differences, whether they diminish as intensity increases or whether they remain constant, one may infer, respectively, the presence or absence of recruitment.

RESULTS AND COMMENT

1. *Can Equal-Loudness Judgments Be Made with Speech Sounds?*—It seemed to us that if one wanted an estimate of recruitment for speech, the most direct attack would be to use speech itself. We found it easy to induce persons with normal hearing to listen to connected discourse at a constant intensity (so far as that condition can be achieved) and to make equal-loudness matches at a variety of sensation levels. The data for two typical normal-hearing subjects are presented in Figure 2*A*. The 45-degree diagonal represents exactly equal loudness for the two ears at every sensation level. It can be seen that with normal-hearing ears the loudness matches do not at any intensity differ significantly from this diagonal.

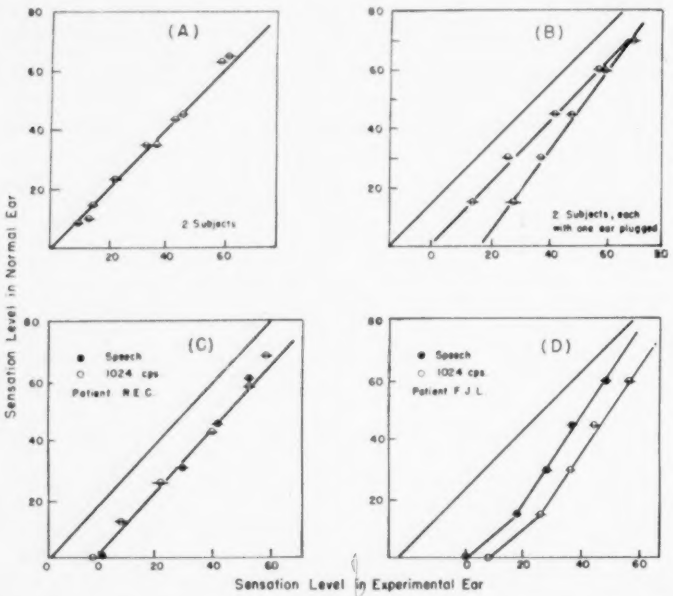

Fig. 2.—Graphs showing recruitment in patients with conductive deafness. (Horizontal line through a point equals 2 standard deviations.) *A* and *B* show precision of loudness matching of speech between two normal ears, with and without artificial obstruction. *C* and *D* show no loss of ability to make precise loudness matches with monaural deafness.

Otologist's notes on Patient R. E. C., a youth 18 years old: Right Eustachian tube was blocked. Drum membrane was normal. Patient had slight cold. Family history of deafness was present. Patient's deafness was probably conductive type, possibly otosclerotic. Radium treatments were prescribed. After eight radium treatments, no improvement was noted.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	15	20	20	35	55	25	50	50
L:	— 5	0	0	5	10	— 5	5	15

Otologist's notes on Patient F. J. L., a youth 19 years old: Family history was negative for deafness. Patient had acute hemorrhagic aero-otitis media with conductive type deafness. Some blood was present in left ear also.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	35	40	45	35	35	40	45	30
L:	15	15	10	5	5	20	20	5

We are thus assured that the inevitable peaks and troughs in the loudness of speech can within the space of a few words be averaged (or disregarded) sufficiently to make equal-loudness judgments possible and that these judgments in untrained subjects may be about as precise as if continuous pure tones were used.

2. What Effect Does Artificial Conduction Deafness Have on Loudness Matching?—Our next step was to ascertain whether partially deafening an ear by artificial means would disrupt equality judgments. Figure *2B* shows data for two typical normal-hearing subjects, each with a warden in one ear. The standard deviations as compared with those of Figure *2A* reveal that no loss whatever in precision is caused by the simulated conduction deafness. It can be noted that as the intensity in the unobstructed ear is increased, the loudness matches follow the 45-degree line in slope, displaced only to the extent of the effect of the ear warden. (In the subject whose curve is shown on the right in Figure *2B*, for whom the warden was a better acoustic seal, the attenuation became less at higher intensities, and thus the matches converged toward the 45-degree line.)

3. How Do Loudness Matches for Pure Tones and for Speech Compare?—The marked success of loudness matching for speech with artificially produced conduction deafness led us to attempt a direct comparison of loudness matching with pure tones and with speech, using patients with monaural deafness.

A. Patients with Pure Monaural Conduction Deafness: Patients were selected who showed a difference of at least 10 db. between the right and the left air-conduction audiograms through the speech frequencies and for whom the diagnosis was pure conduction deafness. Figure *2C* shows a typical case. A graph of loudness matches produced a straight line parallel to the 45-degree diagonal. Two facts will be noted: First, since the graph does not approach the 45-degree diagonal, no tendency is seen for recruitment to occur, and, second, both sets of data, for speech and for 1024 cps, are best fitted by the same straight line.

Another typical case is presented in Figure *2D*. After an initial period of about 18 db. above the worse-ear threshold (during which period the graphs proceed parallel to the 45-degree diagonal just as in Figure *2C*), the graphs change slope and show some tendency to approach the diagonal. It is clear that for this patient also the graphs for speech and for pure tones are entirely similar.

B. Patients with Pure Monaural Perceptive Deafness: Four patients in this category were studied carefully with both pure tones and speech. Four quite different audiograms were obtained: (*a*) flat, (*b*) high-tone slope, (*c*) somewhat dipped in the middle, and (*d*) unequal binaural loss. Recruitment in these cases is shown in Figure 3. With all patients the classic picture of the Fowler phenomenon is obtained (with reservations as to the shape of the curves of best fit, to be discussed later). The graphs leave little reason to doubt that if recruitment occurs for pure tones in the speech region it will also occur (and with almost exactly the same characteristics) for speech itself. Moreover, it is as easy and as precise (note the standard deviations) to measure the one as the other.

An additional point should be noted in Figure 3*C* and *D*. On each of these two patients, two different pure tones were studied through the speech frequencies. It is seen that the data for speech follow an intermediate position among the data for those tones. The same state of affairs is shown even more clearly with the next category of patients, and it may be taken as a general rule, to which we have seen

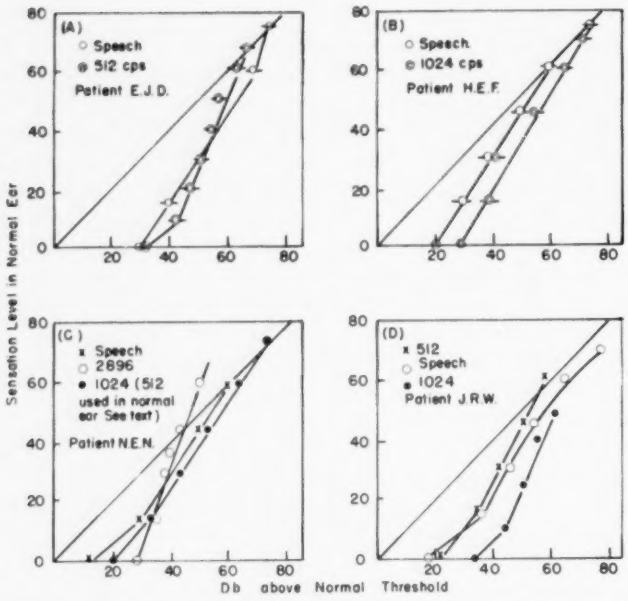

Fig. 3.—Graphs showing recruitment in patients with perceptive deafness.

Otologist's notes on Patient E. J. D., a man 24 years old: There was no deafness in family. Patient had mastoiditis in right ear at age of 10 years. Left ear was affected during the past year. Patient was exposed to diesels and artillery in service. Bone-conduction audiogram and tuning-fork tests denoted cochlear dysfunction. Patient had perceptive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	— 5	0	15	20	10	— 5	5	15
L:	30	30	50	40	40	25	40	50

Otologist's notes on Patient H. E. F., a youth 19 years old: There was no deafness in family. Patient had severe blow on head four years previously and was "completely deaf" for two months. Five of six clinical signs indicated cochlear dysfunction. Patient had perceptive type deafness:

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	5	0	0	0	5	0	20	30
L:	5	10	40	65	70	50	45	35

Otologist's notes on Patient N. E. N., a man 29 years old: There was history of deafness in family. Patient had hearing deficiency for at least 12 years. He was subjected to diesel engine noise for past 10 years. His conduction system was normal. Deafness was of perceptive type.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	0	0	30	15	15	25	45	25
L:	0	5	35	35	50	25	35	40

Otologist's notes on Patient J. R. W., a youth 19 years old: Deafness was present in family. Patient had hearing deficiency for three years. Large adenoids were noted. Bone-conduction audiogram and tuning-fork tests denoted cochlear dysfunction, probably familial. Patient had perceptive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	25	25	25	25	40	55	55	30
L:	45	50	65	65	75	70	75	60

little exception, that the absolute amount and likewise the slope of recruitment for speech will adopt a rough average of the data for pure tones.

C. Patients with Monaural Mixed Deafness: A number of patients in this more readily available category were studied with speech and with a selection of pure tones. Graphs for four typical patients are shown in Figure 4. As would have been predicted from the preceding categories, in mixed deafness also the data for speech are of the same sort as, and are roughly intermediate between the data for pure tones.

What is the Actual Shape of the Recruitment Curve?—The course that recruitment takes as sounds grow from weak to loud has not always been carefully considered. It is often the case that merely the presence or absence of recruitment is all one wants to know. However, a knowledge of whether recruitment for weak sounds proceeds more rapidly or more slowly than for louder sounds can be important clinically and is, moreover, of first importance when we come to consider the origin and nature of the phenomenon.

The first explanation in neurological terms was given by Lorente de Nó.[8] He reasoned as follows:

> If a number of hair cells in the ear or a number of fibres in the cochlear nerve is missing, the tones will appear to be weaker in intensity when near threshold stimuli are used; but if the intensity of the tone is increased, the more strongly activated hair cells or cochlear fibres will be sufficient to saturate, i. e., to excite with the limiting intensity the cochlear fibre or the cells of the cochlear nuclei, so that the cerebral cortex will receive the same number of impulses per second from both ears and will perceive the tone delivered to the diseased ear as strongly as the tone delivered to the normal or less affected ear.

Although recruitment has not always been exhibited with cases of known neural deafness, there can be no question but that with lesions in certain areas of the auditory system Lorente de Nó's explanation in terms of neural summation and occlusion must be correct. Its only shortcoming is that it does not predict the shape of the curve of recruitment.

Another type of explanation is given by Steinberg and Gardner.[9] Their explanation, moreover, makes a prediction as to the shape of the recruitment curve and can, therefore, be put to experimental test. They assume with Fletcher [10] that loudness is related to the total number of nerve impulses per unit time. Now, if an ear is deficient in number of nerve fibers, a certain amount of loss of loudness is expected and this loss of loudness remains constant throughout all intensity ranges.

These ideas are symbolized in Figure 5. Curve 1, the 45-degree diagonal, represents equal growth of loudness in two normal ears, as in previous figures. Curve 2, on the other hand, represents the growth of loudness in an ear with a constant loss of about 100 millisones, or loudness units (a tone of 1000 cps at 40 db. is said to have a loudness of 1,000 millisones). It should be noted that this ear will be 20 db. defective at threshold, since for the normal ear a loudness of 100 units is reached only when the sound is about 20 db. over threshold; but when the intensity is raised

8. Lorente de Nó, R., in discussion on Fowler.[7]

9. Steinberg, J. C., and Gardner, M. B.: The Dependence of Hearing Impairment on Sound Intensity, J. Acous. Soc. Am. **9**:11-23, 1937.

10. Fletcher, H.: Newer Concepts of the Pitch, the Loudness, and the Timbre of Musical Tones, J. Franklin Inst. **220**:405-429, 1935.

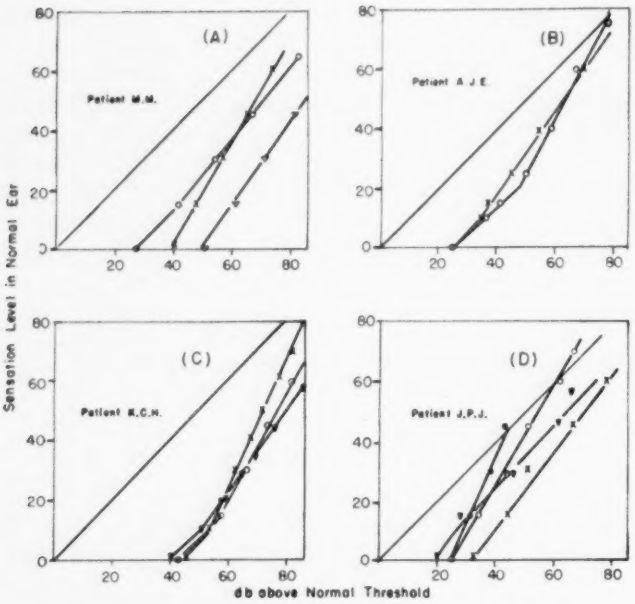

Fig. 4.—Graphs showing recruitment in patients with mixed deafness.

Otologist's notes on Patient M. M., a woman 36 years old: There was no deafness in family. Patient had low-frequency tinnitus and feeling of "fulness" in left ear. She had had many colds. Drum membranes were slightly retracted. Granular adenoids were present, which had been treated with radium. Bone-conduction audiogram indicated some cochlear dysfunction. Patient had mixed type deafness, largely conductive.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	0	0	5	0	0	0	25	15
L:	45	45	45	35	35	25	45	40

Otologist's notes on Patient A. J. E., a youth 19 years old: Family history and personal history were apparently completely negative for deafness. Patient was never subjected to loud noises or had ear trouble. He thought he heard as well with left ear as with right and noticed no change in hearing ability. Appearance of both ears was normal. Bone-conduction audiogram showed some cochlear dysfunction in left ear. Both ears were possibly otosclerotic. Patient had mixed type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	20	20	20	15	15	5	20	40
L:	45	45	45	30	30	30	45	60

Otologist's notes on Patient K. C. H., a man 25 years old: There was no deafness in family. Patient was subjected to loud 5-in. gun blast five years previously. Drum membranes were clear. Inflation of middle ear was slow. Radium was applied to nasopharynx. Bone-conduction audiogram showed some loss. Patient had mixed type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	35	40	45	35	30	25	30	25
L:	0	5	0	0	5	0	15	30

Otologist's notes on Patient J. P. J., a man 18 years old: Mother had monaural deafness. Hearing in patient's right ear was reduced since scarlet fever nine years previously. Head colds reduced acuity in right ear. Right eardrum membrane was perforated five years previously. Right ear showed purulent discharge with granulations on drum membrane. Adenoids were large. Patient had mixed type deafness.

(Legend continued on opposite page)

to 40 db. the loudness for the normal ear is 1,000 units. Thus, relatively, the ear in question is only 10% defective, and by the time the intensity reaches 73 db. the loudness for the normal ear is about 10,000 units, so that the defect in question is less than 1%—it cannot, indeed, be detected. Similarly, Curves 3 and 4 symbolize the loudness growth for ears with constant loudness losses of 1,000 and 4,350 millisones, respectively.

We see in Figure 5, then, that the predicted curves of recruitment allow us to put Steinberg and Gardner's loudness-loss explanation to a test. But before we bring data to bear on this or other theories of recruitment, it will be well to present a classification of the types of recruitment curve actually found. In the first place, there is no necessity for all cases of recruitment to have the same cause—some cases, for instance, might be caused by increased labyrinthine pressure; other cases, by temporary damage to the hair cells; still others, by damage of first-order nerve fibers, etc. In the second place, it is not necessary that all recruitment curves should take the same shape. We have, indeed, distinguished four main types: first, asymptotic recruitment, with all points fitted by a single curve asymptotic to the 45-degree diagonal; second, straight-line recruitment, with all points fitted by a single straight line; third, delayed recruitment, with all points fitted by two straight lines, and fourth, delayed plus asymptotic recruitment, combining the features of the first and third types. We will discuss these types in order.

Type 1, Asymptotic Recruitment: The existence of this type, early described and given a plausible explanation by Steinberg and Gardner, has overshadowed other possible types. Indeed, it has become common practice to draw an asymptotic curve through any set of recruitment data without regard to significant experimental divergences from that line. Let us check the suitability of an asymptotic curve and thereby bring it to bear on the loudness-loss explanation of recruitment, by comparing one of the predicted curves of Figure 5 with data from an actual patient.

The crosses in Figure 5 represent loudness matches for a patient of Fowler,[6] who had a 60 db. loss at 2048 cps. The data do not very well follow the course of Curve 4, but, on the other hand, a moment's thought will show that actual data could never fit such a curve. For a good fit to occur, it would be necessary that the

---

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	15	20	25	30	35	15	25	70
L:	5	5	0	5	5	— 5	0	15

All tests summarized in Figure 4*D* were made same day as audiogram shown above. Two weeks later, after acute infection had subsided, another audiogram was made:

	256	512	1024	2048	2896	4096	5792	8192
R:	10	10	10	20	35	15	30	50

Distinct improvement at and below 2048 cps confirmed notion of a conductive component in deafness for these tones shown in first audiogram. On the other hand, perceptive component at higher frequencies did not materially improve. This patient thus presented a unique opportunity to examine what happens to speech recruitment when only a portion of the spectrum is available. This was easily done with filtering, as follows: A 1000 cps low-pass filter, which cuts off all higher frequencies at 45 db. per octave, was inserted in speech circuit, and loudness matches again required. In this case no recruitment at all occurred for first 18 db. above worse ear threshold, after which recruitment did indeed begin and eventually became complete. This course is, however, to be contrasted with that for unfiltered speech as shown in Figure 4*D*, in which 12 db. of recruitment, rather than none, occurred within the first 18 db. above worse ear threshold.

effects of a 2048-cps tone not spread to other frequency regions on the basilar membrane and that the patient be 60-db. deaf for 2048 cps but perfectly normal for all other frequencies. Of course, neither condition can be met. One has to consider that any pure tone of 60 db. or more intensity will set into activity extensive portions of the basilar membrane, in addition to the restricted area for which at that frequency the membrane is most sensitive, and that these outlying portions will contribute their share to the total loudness. Furthermore, no patient has an audiogram perfectly perpendicular on either side of 2048 cps. Consequently, as the activity on the membrane spreads with more and more intense sounds, the status of hearing at other frequencies must be utilized in calculating the loudness of these sounds.

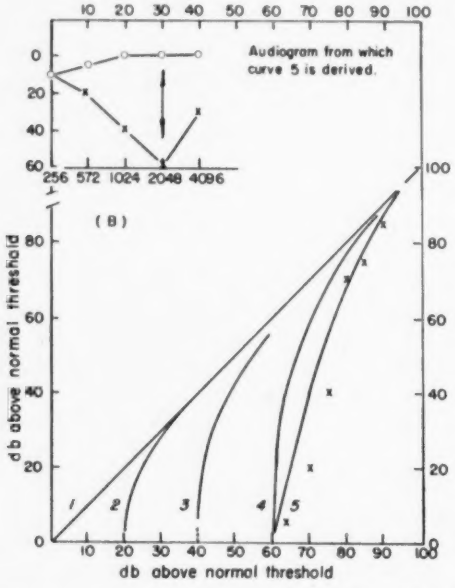

Fig. 5.—Recruitment as expressed in sensation level. (For explanation of Curves 1 to 5, see text.)

The fundamental concepts for calculating the loudness of a sound in a partially deafened ear have been provided by Fletcher and Munson.[11] The masking audiogram of the sound for a normal ear must first be known. (For a steady-state complex sound, this can be determined by anyone with an audiometer; for pure tones a selection of frequencies and intensities is given by Wegel and Lane [12] and summarized in a more readily available source.[13] From the masking audiogram one may infer an activity pattern on the basilar membrane. Each sound at each intensity will, of course, have a different activity pattern. Fletcher and Munson, by a series of reasonable assumptions, transform this activity pattern into a loudness

11. Fletcher, H., and Munson, W. A.: Relation Between Loudness and Masking, J. Acous. Soc. Am. **9**:1-10, 1937.

12. Wegel, R. L., and Lane, C. E.: The Auditory Masking of One Pure Tone by Another and Its Probable Relation to the Dynamics of the Inner Ear, Physics Rev. **23**:266-285, 1924.

13. Fletcher, H.: Speech and Hearing, New York, D. Van Nostrand Company, 1929.

pattern and thus enable one to find the loudness density, in millisones, contributed by each 1% of the membrane. The total loudness of any sound may then be found by adding up (graphically) the millisones contributed by each unit length. They provide a convenient chart upon which one may enter the masking audiogram directly, without any conversion to loudness density, and find very quickly the loudness in millisones of that sound for the normal ear. To find the loudness of that sound for a deafened ear, one has only to plot the patient's audiogram on the same chart and find the difference in millisones between the curves. This procedure is illustrated by a general case in Figure 6 and provides a very handy way for the clinician to estimate what the loudness of certain types of sound will be for particular patients.[14]

When, furthermore, this method is applied to the patient with the audiogram shown in Figure 5, the result is as shown in Curve 5. Obviously, the fit of Curve 5 is much better than that of Curve 4 to the actual data and represents a more

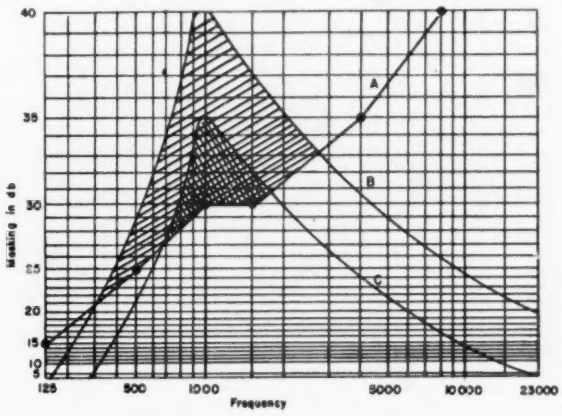

Fig. 6.—Procedure for calculating loudness loss due to deafness. Curve *A* represents audiogram; *B*, masking due to narrow-band noise centering at 1000 cps; *C*, masking due to same noise, 5 db. weaker. Entire area represents 37,000 millisones; double hatched area represents loudness of weaker noise as heard by ear of Curve *A*; total hatched area represents loudness of louder noise.

realistic formulation of the problem. However, if the sensation levels of Curve 5 are converted to millisones, the original loudness loss (at threshold) of 4,350 millisones would be seen not to remain constant at 4,350 as with Curve 4 but, instead, rise to a loss of 6,975 within 10 db. and to a loss of 11,045 millisones within 20 db. above the deaf threshold.

What, then, becomes of the constant loudness-loss explanation of recruitment, since in Curve 5 a continuous increase in loudness loss, rather than a constancy,

14. The extension of the general method to the case of the deafened patient was first made by Steinberg and Gardner,[9] who provide a slightly different chart in which the coordinates are distance in millimeters from the helicotrema and loudness density per millimeter on the basilar membrane. All in all, however, the chart of Fletcher and Munson[11] is more suited to the form in which the clinician's data are usually cast.

is exhibited? The discrepancy between Curves 4 and 5 militates against the constant loudness-loss theory, though it throws little light on what other theory may be correct.

It is one question whether a particular set of recruitment data exhibit an asymptotic form best fitted by the technique of Fletcher and Munson, and another question whether the best-fitting line is asymptotic at all. As a matter of fact, we

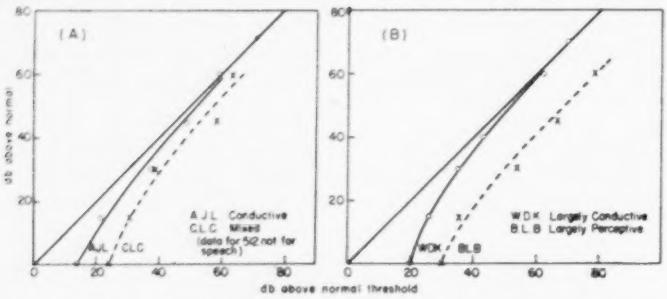

Fig. 7.—Graphs showing asymptotic recruitment for speech.

Otologist's notes on Patient A. J. L., a youth 19 years old: There was no deafness in family. Cyst was removed from left side of neck at age of 4 years, with discharge from left ear as result. Marked retraction of left drum membrane, with wax. Hypertrophied adenoids were noted, otherwise findings were normal. Shape of all speech-articulation data was quite normal. Bone conduction was normal through 1024. Patient noticed left ear being "weak" for five to six years; he does not think condition is progressive now. Otologist's impression was conductive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	0	0	5	5	0	5	0	5
L:	20	20	25	15	5	15	40	20

Otologist's notes on Patient C. L. C., a youth 17 years of age: There was no deafness in family. Patient had abscess in right ear at age of 8 years. Adenoids were hypertrophied, and tubes were not patent. Otherwise conduction system appeared normal. Speech-articulation curves were normal in shape. In Naval Training Station patient was on firing range and had ringing in ears for a "few days." Bone conduction was nearly normal. Otologist's impression was conductive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	30	40	10	15	20	5	20	20
L:	10	10	10	5	5	15	15	25

Otologist's notes on Patient W. D. K., a youth 20 years old: Uncle was hard of hearing in both ears. Brother was hard of hearing, but relieved on treatment by otologist. At time of examination patient had bad head cold and stated only time he had hearing difficulty was when he had a cold. Bone conduction was normal except at 4096. Speech-articulation curves were of normal shape or steeper than normal. Otologist's impression was conductive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	10	25	20	20	20	25	20	15
L:	5	15	10	0	5	15	20	10

Otologist's notes on Patient B. L. B., a youth 19 years old: Brother had monaural hearing defect. Patient had earache after swimming, three years previously. Drum membranes were normal, moderately retracted. Appearance of conduction system was otherwise normal. All speech-articulation curves were of perfectly normal shape. There was 25 db. loss in right ear at 1024 and 2048 by bone conduction. Otologist's impression was mixed type deafness, largely perceptive at speech frequencies.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	35	35	35	25	10	15	10	15
L:	5	5	10	0	5	0	0	0

have found considerably fewer asymptotic curves for our patients than would have been predicted from the literature. A case clearly appears in the data for 512 cps in Figure 4*D*. Four more patients are represented in Figure 7. It is our experience that this type of recruitment is in the minority.

It should be pointed out that the data from the four ears with recruitment presented by Steinberg and Gardner (their Figures 2 and 12) are fitted by asymptotic curves, though apparently they are also fitted equally well by straight lines; unfortunately, graphs of the data for these four ears are too limited in size to make possible a rigid statistical comparison of lines of best fit. On the other hand, an asymptote is unquestionably the best fit for the graphs of a number of patients in our Figures 4, 7, 8, 9, and 10.

The conditions which will produce asymptotic recruitment are still unclear. It usually appears in Ménière's disease (though in Case 3 of Figure 10 it does not appear, nor does it appear for the frequencies 250, 1000, 2000, and 8000 cps for Patient 27 [our Fig. 9], the only patient with Ménière's disease cited by de Bruïne-Altes[15] [p. 100]; it usually appears in traumatic perceptive deafness

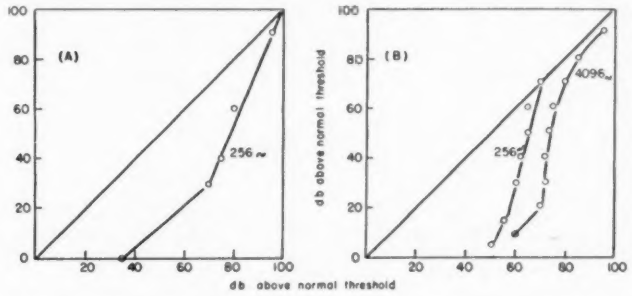

Fig. 8.—Graphs showing recruitment data from Fowler (Footnotes 6 and 7). *A* is from Fowler,[6] p. 739, and *B* is from Fowler,[7] p. 216.

[Patients 41, 43, 45, and 46 in Figure 9], and it usually does not appear in blast deafness [Patients 37 and 39 in Figure 9]). It is certainly significant, as has been maintained for some years, that asymptotic recruitment is not usually found in patients with pure conductive deafness (though Patients A. J. L. and W. D. K. in Figure 7 had largely conductive deafness). On the other hand, it is equally significant that not all patients with perceptive deafness exhibit an asymptotic curve.

At this point it should be mentioned that asymptotic recruitment is invariably the rule with simulated monaural deafness from a noise mask. Figure 11 unequivocally shows curves of Type 1 in the recruitment data for a normal-hearing subject in a monaural noise mask of 20, 30, or 40 db. effectiveness. Since this sort of curve is not by any means always encountered in perceptively deaf ears, it follows that the introduction of a noise mask does not always truly simulate what is often rather loosely called "nerve deafness." Factors other than the "line-busy" effect and inhibition of various sorts (operating in the noise-masking

15. de Bruïne-Altes, J. C.: The Symptom of Regression in Different Kinds of Deafness, Thesis, Gröningen, Holland, 1946.

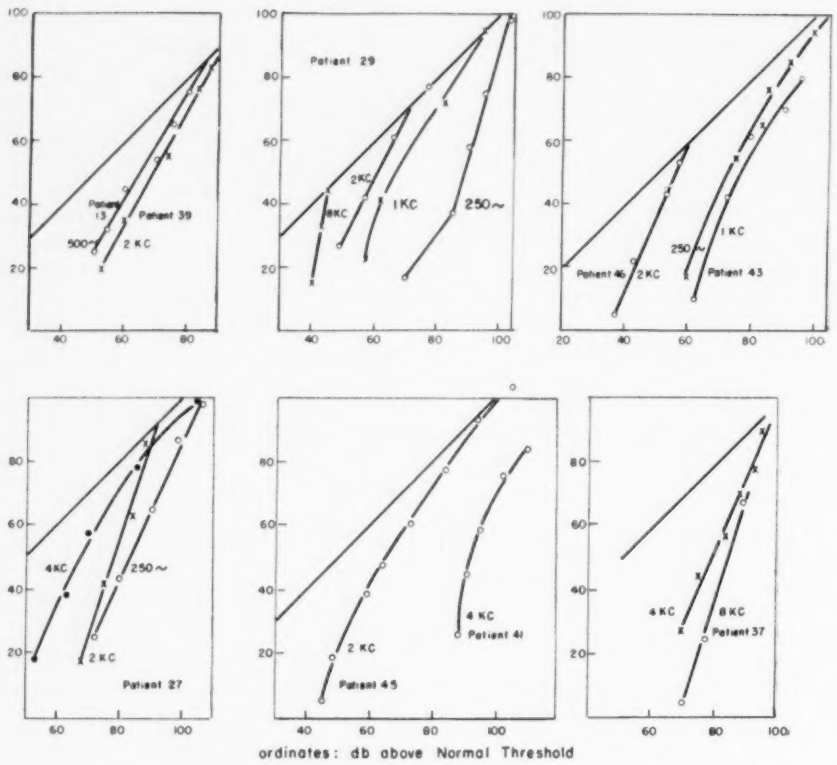

Fig. 9.—Recruitment data from de Bruïne-Altes.[15]

Following histories are quoted verbatim from de Bruïne-Altes. Some awkwardness in rhetoric is occasioned by the translation.

Patient 13, woman aged 20: progressive hereditary perceptive deafness:

	125	250	500	1000	2000	4000
R:	5	10	25	70	95	110
L:	10	25	50	75	110	120

Patient 27, woman aged 41: Ménière's syndrome:

	125	250	500	1000	2000	4000	8000
R:	55	60	65	65	70	50	55
L:	5	10	— 5	5	20	10	20

Patient 29, man aged 36: polyneuritis cerebralis menieriformis, involving sixth through tenth nerves. A week after onset of acute infectious jaundice patient had peripheral paralysis of seventh nerve on left, accompanied with pain in left eye (keratitis with lagophthalmos) and the left half of the face, tinnitus on left, dysphagia, and hoarseness. He did not have an attack of dizziness, only a light-headed feeling. Hearing on left, which had been less acute than on right for one year, markedly decreased. Four weeks later his hearing was extensively examined. Tumor was ruled out by substantial return of hearing six months later.

	125	250	500	1000	2000	4000	8000
R:	5	5	10	15	10	0	5
L:	65	60	65	60	45	25	25

Patient 37, youth aged 19, gun deafness (small arms):

	125	250	500	1000	2000	4000	8000
R:	5	5	5	15	15	70	65
L:	— 5	5	— 5	10	10	—10	— 5

(Legend continued on opposite page)

situation) must, of course, be fundamental in certain cases of inner-ear disturbance. It is clear that one day such catchall phrases as "nerve deafness" or "perceptive involvement" will be replaced by more specific statements.

Type *2*, Straight-Line Recruitment: Five of the eight patients in Figures 3 and 4 yielded one or more curves of straight-line recruitment. Curves of two more patients are shown in Figure 12. Nine curves of straight-line recruitment are shown from six patients of de Bruïne-Altes (Fig. 9). The involvements of these 13 patients range from pure conductive to pure perceptive deafness. Evidently straight-line recruitment does not relate directly to the usual categories of deafness.

One must not confuse the regularity of straight-line recruitment with the constant loudness-loss theory. What is constant with this type of recruitment is the regular reduction in the sensation-level difference between the ears. In the graph in Figure 3*B*, for example, showing data on speech perception for Patient H. E. F., the initial deficit of 20 db. in the affected ear was overcome at the rate of 3.3 db. for every rise of 10 db. sensation level in the normal ear. In other words, the sensation level of the experimental ear increases at one-third again the rate of a normal ear.

Patient 39, man aged 45, gun deafness (small arms):

	125	250	500	1000	2000	4000	8000
R:	15	15	15	15	15	65	35
L:	15	25	15	10	45	65	50

Patient 41, man aged 36, explosion trauma:

	125	250	500	1000	2000	4000	8000
R:	30	25	25	20	25	15	30
L:	15	25	30	25	30	90	80

Patient 43, man aged 36, commotio labyrinthi from blow on head with stick. Two years before examination, patient was thrashed with a stick in a concentration camp. "He then received a blow with a stick on right side of his head, so that he staggered. After this maltreatment he was somewhat dazed for three days. Since then hearing acuity of his right ear has been defective. This remains stationary. He is also troubled with attacks of dizziness." These attacks recurred usually within a fortnight, irregularly, and lasted 15 minutes to a few days.

	125	250	500	1000	2000	4000	8000
R:	40	55	55	55	70	75	80
L:	5	10	15	20	45	35	35

Patient 45, man aged 34, explosion trauma with commotio cerebri because of direct head injuries. "Patient was driving a car when at a distance of 3 meters left of the car a bomb exploded. He went spinning through the air. He lost consciousness for a few moments. Afterward he was somewhat dizzy, slightly deafened, and troubled with tinnitus in right ear. Under left zygomatic arch a splinter of the bomb had penetrated, which had struck at the level of the jaw joint. Eight days after the accident he was audiometrically examined. The hearing had already slightly improved."

	125	250	500	1000	2000	4000	8000
R:	0	0	5	10	10	60	35
L:	10	0	5	20	50	70	65

Patient 46, man aged 29, explosion trauma with commotio cerebri from direct head injuries. "About five weeks before this audiometric examination a bomb fell at a distance of 4 meters on the left of this patient. There was an excessive noise. He was unconscious for a quarter of an hour. A splinter of the bomb in his left auricle was removed the same day. . . . For one day he was dizzy without nausea or tinnitus. After a month the traumatic otitis media was cured."

	125	250	500	1000	2000	4000	8000
R:	0	10	10	10	10	10	10
L:	15	25	25	25	35	50	55

Type 3, Delayed Recruitment: A common type of recruitment is that in which for the weaker sensation levels in the experimental ear no recruitment is shown, then (with increasing intensity) recruitment appears and may even become complete. It is interesting that the first two cases of unilateral nerve deafness for

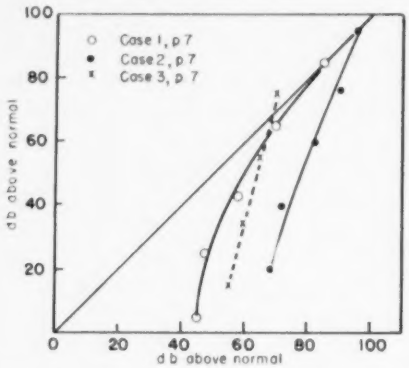

Fig. 10.—Recruitment data (Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Proc. Roy. Soc. Med. **41**:516-526, 1948).

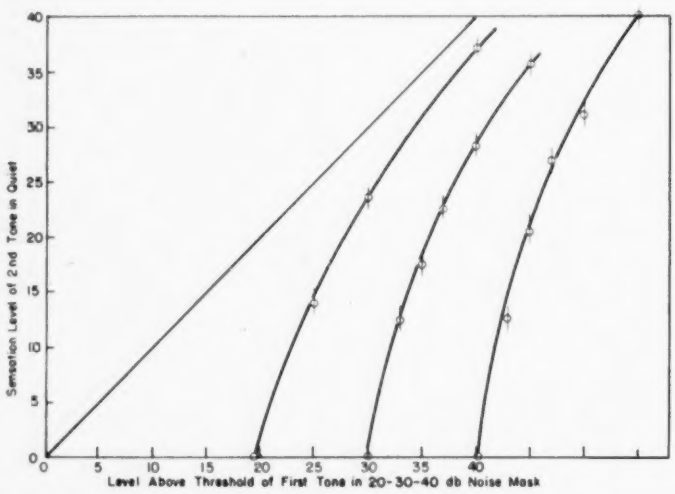

Fig. 11.—Pseudorecruitment in simulated deafness (noise masking). Frequency was 1000 cps. Both tones were 0.25 second long, separated by 1 second of silence; second tone, in quiet, was adjusted to match by method of constant stimuli. Vertical lines through circles indicate 2 standard errors.

which recruitment data were published, by Fowler,[16] showed this delay feature (Fig. 8). It is mentioned also by Watson [17] (p. 79).

16. Footnotes 6 and 7.

17. Watson, L. A., and Tolan, T.: Hearing Tests and Hearing Instruments, Baltimore, Williams & Wilkins Company, 1949.

Six patients in Figures 2 to 4 yielded one or more delayed recruitment curves (Figs. 2*D*, 3*A*, 3*C*, 3*D*, 4*B*, 4*C*). Curves of seven more patients are shown in Figures 13 and 14. Hearing difficulties in these 13 patients include such diverse categories as pure conductive, pure perceptive, and mixed deafness in a variety of proportions from pure perceptive with slight head cold to largely conductive with perhaps some perceptive involvement.

Several explanations come to mind for the delay feature. The most obvious possibility is that the so-called normal ear of the patient may be partially deaf but to a less degree than the experimental ear. In that case, at weak intensities at which both ears may have recruitment at the same rate, loudness matches between the ears would show no differential rise for the experimental ear; only when the intensity rises so that the mild deficiency of the better ear is overcome would recruitment seem to set in for the experimental ear. In some examples of delayed

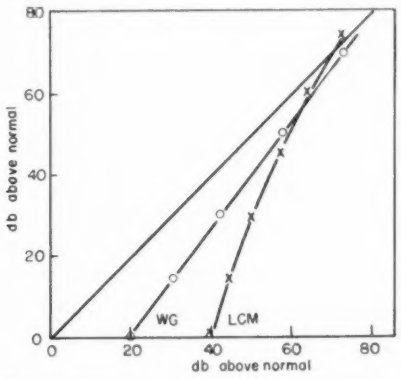

Fig. 12.—Straight-line recruitment for speech in mixed (Patient W. G.) and conductive (Patient L. C. M.) deafness.

Otologist's notes on Patient L. C. M., a man 21 years old: Family history was negative for deafness. Patient had fungus in left ear six months before, now cured; ears itched and discharged after swimming. Drum membranes were clear and intact. History, shape of audiogram, air conduction-bone conduction differential, and shapes of speech-articulation curves indicated pure conductive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	0	0	0	0	0	15	10	— 5
L:	40	50	40	30	35	50	45	50

Otologist's description of Patient W. G., a man 27 years old: Father was hard of hearing in one ear. Patient often had some deafness in left ear after gunfire. He noticed hearing in left ear to be somewhat defective for past three years and believed loss was not progressive. Eustachian tubes were blocked; both drum membranes were retracted; large adenoids were present. Speech-articulation curves were of normal shape. Test with tuning fork at 512 indicated conductive impairment. Speech at equal loudness in the two ears had a "quite different tonal effect." Patient had mixed deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	5	0	0	5	30	75	80	75
L:	30	30	25	45	40	55	70	30

recruitment this is undoubtedly the case. For example, in Figure 3*D*, there is a delay of about 10 db. at 1024 cps before recruitment sets in. In this patient, with perceptive type deafness, probably familial, the hearing in the experimental ear

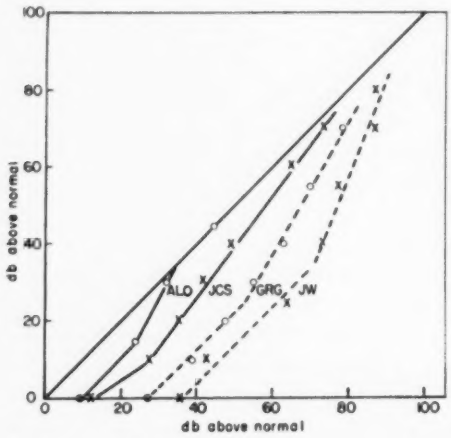

Fig. 13.—Delayed recruitment for speech in mixed, largely perceptive (Patient A. L. O.), perceptive (Patients J. C. S. and G. R. G.), and mixed, largely conductive (Patient J. W.), types of deafness.

Otologist's notes on Patient J. C. S., a youth 20 years old: Family history was negative for deafness. Patient first noticed defect two months previously. He thought at time it was due to a cold. He attended electronics school for nearly a year just prior to examination. He noticed that he had to remove headphones many times, as sound from a signal generator was too loud and caused ringing in ears. At present no tinnitus is present. Eustachian tubes are patent; meatus, drum membrane, and ossicles are normal. Speech-articulation curves are of normal shape. So far as can be determined, both ears probably have recruitment, and curve in Figure 13 shows difference in recruitment rates of the two ears. Patient had perceptive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	20	15	15	45	40	20	25	60
L:	25	25	50	35	40	40	60	70

Otologist's notes on Patient A. L. O., a man 21 years old: Family history was negative for deafness. Patient had no tinnitus but severe head cold was present. Otherwise no abnormality was noted. Articulation curve for female whispered voice was of normal shape; curves for male spoken voice were of steeper than normal slope. Otologist's impression was mixed deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	10	10	5	15	25	35	30	15
L:	25	30	20	25	40	55	80	40

Otologist's notes on Patient G. R. G., a man 21 years old: Family history was negative for deafness. Two months before examination patient was subjected to 600 rounds of 40 mm. firing; ringing remained in right ear for more than two days. Appearance of conductive system was normal. Bone-conduction audiogram was depressed. Speech-articulation curves were of normal shape. Patient had perceptive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	20	25	45	60	70	80	80	80
L:	15	15	10	15	20	45	40	40

Otologist's notes on Patient J. W., a man 21 years old: There was history of otosclerosis in family. Patient first noticed hearing difficulties one and a half years previously. He believed loss was progressing. Continuous ringing was present in left ear. Patient fired many rounds of 40 mm. shells. Both drum membranes were red; hypertrophied adenoids were present, with tubular obstruction, especially in left ear. All speech-articulation curves were of normal shape. Otologist's impression was conductive type deafness, with probably some perceptive involvement.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	5	5	5	10	10	5	0	0
L:	60	60	55	40	50	55	50	35

was decreased 65 db. and that in the better ear only 25. In a patient with mixed deafness (Fig. 4*B*), the hearing in the experimental ear was reduced 45 db. and in the better ear, 20 db.; recruitment set in after a delay of about 25 db.

A similar situation existed for the patient in Figure 8*B*; according to the data for 256 cps, a deafness of 5 db. was present in the better ear and a delay of about 5 db. occurred. A correspondence between a 10 db. deafness in the better ear and a 10 db. delay occurred likewise at 4096 cps, according to the data presented.

On the other hand, recruitment of the better ear cannot explain some curves. For example, in Figure 3*A* a delay of 10 db. was observed, yet the better ear had no loss whatever at 512 cps. Similarly in Figure 4*C*, at both 512 and 1024 cps there were delays of about 10 db., yet the better ear was entirely normal. The

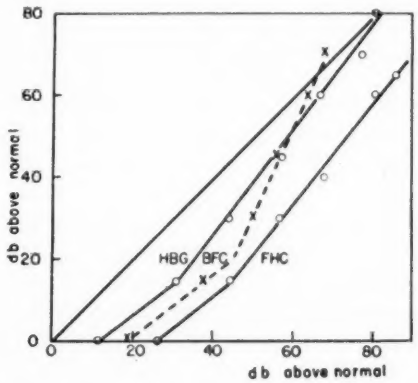

Fig. 14.—Delayed recruitment for speech in mixed type deafness. Otologist's notes on Patient B. F. C., a youth 18 years of age: There was no deafness in family. Patient had earaches in both ears at age of 6 to 7 years. Appearance of conductive system was normal. Bone conduction was normal. Otologist's impression was slight conduction deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	5	5	0	− 5	0	5	0	0
L:	15	15	15	5	10	5	0	0

Otologist's notes on Patient H. B. G., a youth 19 years old: There was no deafness in family. Patient went to sick bay three months prior to examination, complaining of sore throat and impaired hearing; he was told Eustachian tubes were blocked. At time of present examination, sounds seemed "muffled" in right ear. Patient had high-frequency ringing in right ear most of the time. Bone-conduction loss coincided with air-conduction loss. Otologist's impression was mixed deafness, largely perceptive.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	10	15	45	55	55	55	45	25
L:	10	15	20	15	15	15	20	5

Otologist's notes on Patient F. H. C., a youth 18 years old: There was no deafness in family. Patient had bad earache and infection in right ear about five years previously. About a month prior to examination, he reported to sick bay to have his right ear washed out, whereupon "hearing improved." Appearance of conduction system was now normal. Speech-articulation curves were of normal shape. Bone conduction was nearly normal. Otologist's impression was conductive type deafness.

Audiogram:

	256	512	1024	2048	2896	4096	5792	8192
R:	25	20	30	15	35	55	35	30
L:	5	0	0	− 5	0	15	5	− 5

same conclusion can be drawn from the case in which the better ear is partially defective and yet no delay occurs. For example, Patient 27 (Fig. 9) showed no delay in recruitment at 250 cps, even though there was a deafness of 20 db. in the better ear according to both air-conduction and bone-conduction measurement. We conclude, then, that one possible, but not the only possible, cause of delay is mild recruitment of the better ear.

Another possible cause of delay is an admixture in the same ear of so-called conductive and perceptive components. Steinberg and Gardner had reasoned that in such a case the perceptive component would cause immediate recruitment but the conductive component would prevent its being complete. This reasoning is supported by cases of mixed deafness in which asymptotic recruitment is, indeed, incomplete as evidenced by the retention by the better ear of some of its superiority at extreme intensities (Patient 27 at 4096 cps and Patient 41 in Figure 9). However, we have no reason whatsoever to suppose that in some cases it would not work the other way; in other words, the recruitment would be apparent only after the stimulus was so intense that the conductive deficiency was overcome. Patient 29 in Figure 9 showed no delay except at 250 cps. But we see from the air-conduction and bone-conduction audiograms that this patient at that frequency only had a 25 db. conduction deafness—enough, on the basis of our supposition, to explain the 20 db. delay.

It is perhaps possible to explain delayed recruitment with unequal binaural loss or with mixed deafness in the above terms, but one must be prepared as well for such cases as that of Patient E. J. D. at 512 cps (Fig. 3*A*). Here, with the better ear perfectly normal and in pure perceptive deafness, a delay of 10 db. nevertheless occurred. An explanation can be straightforward if it is assumed that cochlear elements were damaged so that the threshold was shifted 10 db., but that some of these elements would be fired if the stimulus intensity were raised another 10 db., and still other damaged elements would respond if the intensity were further increased. (These assumptions do no violence to our conception of the distribution of sensitivity of fibers to various intensities, conceptions gained from microelectrode studies.) In summary, as the intensity is raised from the normal threshold at first 10 db. deafness is seen; then for 10 db. a period of no recruitment (i. e., delay) appears; finally, at 20 db. and higher over the normal threshold more and more partially damaged fibers respond and recruitment occurs until it becomes more or less complete. Thus, delayed recruitment has a third possible explanation.

Type 4, Delayed plus Asymptotic Recruitment: The type of curve produced here should be self-explanatory. It is a matter for further experimentation whether this curve exists as a distinct type. We have observed it in a clear-cut fashion for only one patient (Fig. 3*D*, curve for speech). It does not appear in the monograph of de Bruine-Altes, though it does appear in Fowler's work (Fig. 8*B*).

Delayed plus asymptotic recruitment has a logical explanation, of course, in that the delay feature is caused by any one of the three possibilities mentioned above (the two cases cited both had conductive involvements), while the asymptotic feature is the classic picture of perceptive involvement. It is more than likely that this type reveals no new facts about the nature of recruitment; nevertheless, it may be set apart as an aid in organizing the data.

5. *Does the Normal Ear Exhibit Any Analogue of Recruitment?*—Since recruitment is an abnormal growth of loudness, it cannot by definition prevail in the normal ear. Nevertheless, it is interesting to note that in the normal ear the growth of loudness can be affected by a variety of conditions. We list here the effects of frequency, of fatigue, of masking, and of air vs. bone conduction.

A. Analogue of Recruitment as a Frequency Effect: Differing growths of loudness for different frequencies is, of course, inherent in all equal-loudness contours, but for the present discussion another way of plotting the data is instructive. If, for example, one compares 100 and 1000 cps with respect to the energies necessary to achieve equal loudnesses, a plot of the data (Curve 3 in Fig. 15) results which looks quite similar to a typical recruitment curve. Evidently some detail in the structure or function of the normal ear affects the loudness function from frequency to frequency and from intensity to intensity, and, if this detail were

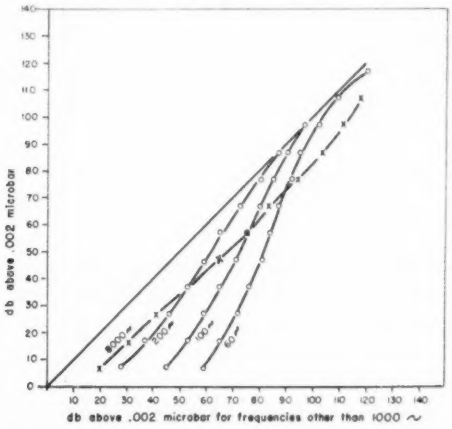

Fig. 15.—Graph showing analogue of recruitment in normal ear (loudness matching of different frequencies). Data are from Fletcher and Munson.[1]

known, it might throw light on the changes in the loudness function of a pathological ear.

There is, indeed, a factor in the physics of the ear which does vary with frequency, namely, the acoustic impedance, or the ratio of the sound pressure to the volume displacement. If certain physical conditions are met, response to low tones is disproportionately depressed; if other conditions are met, response to high tones is disproportionately depressed.

The immediate value of these concepts has been shown by Mygind,[18] Rasmussen,[19] Zwaardemaker,[20] Van Dishoeck and de Wit,[21] Dederding,[22] Johansen,[23]

18. Mygind, S. H.: Further Labyrinthine Studies, Acta oto-laryng., Supp. 68, pp. 53-80, 1948.

19. Rasmussen, H.: Studies on the Effect upon the Hearing Through Air Conduction Brought About by Variations of the Pressure in the Auditory Meatus, Acta oto-laryng. **34**:415-424, 1946.

20. Zwaardemaker, H.: Akustische Eisenbahnsignale und Gehörschärfe, Ztschr. Ohrenh. **28**:33-41, 1896.

(Footnotes continued on next page)

Lüscher,[24] and Veckmans,[25] who have shown that a variety of changes in the mass and/or compliance of the eardrum, ossicular chain, and cochlear structures may produce characteristic shapes of audiogram. Mygind was the first to call attention to the usefulness of the impedance formula in considering these facts.

These ideas become clearer if one uses the familiar electrical analogue: Intensity $= \frac{\text{Energy}}{\text{Resistance}}$. In acoustic terms this may be rewritten: Loudness $= \frac{\text{Energy}}{\text{Impedance}}$ or Loudness $\times$ Impedance $=$ Energy.

We turn to Figure 15 once more and observe that it takes about 20 db. more energy to reach the threshold at 200 cps than at 1000 cps. Now by definition, at the threshold loudness is constant; the 20 db. difference in energy, therefore, can be ascribed only to a difference in impedance. But, as Veckmans insists, in the case of equal-loudness matches at higher intensities one finds the energies at 200 and 1000 cps progressively different, so that by the algebra of the equation Loudness $\times$ Impedance $=$ Energy the impedance must necessarily be varying with energy and in a nonlinear fashion. As he says:

> In order that the increments of energy be different in the two [frequencies], the multiplier [Impedance] must undergo a variation of the same degree [as the Energy], such that Impedance grows relatively less for increments of Energy at 125 cps than for increments of Energy at 1000.
>
> In more familiar terms, when sound energy must overcome greater resistance in order to enter the cochlea—as is the case for low tones with respect to 1000 cps—the pathway becomes from that moment relatively easier and less resistant.

In support of this notion of varying impedance, Veckmans briefly cites experiments with subjects able to contract their intra-aural muscles voluntarily and with a subject having paralysis of the nerve supply to the tensor tympanum; in both of these cases a frequency effect on loudness of low vs. high tones was seen, both in frequency and in intensity.

Veckmans does not give quantitative data to support his theory nor is his sequence of ideas entirely convincing; nevertheless, his theory that high impedance at threshold results in relatively rapid recruitment would account for the low-tone vs. high-tone difference and is at least open to test. The theory furthermore suggests that a heightened impedance is the cause of much recruitment found in clinical material. For example, loudness balances between air-conduction and

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21. Van Dishoeck, H. A. E., and de Wit, G.: Loading and Covering of the Tympanic Membrane and Obstruction of the External Auditory Canal, Acta oto-laryng. **32**:99-111, 1944.

22. Dederding, D.: Clinical and Experimental Examinations in Patients Suffering from Morbus Ménièri, Including Study of Problem of Bone Conduction, Acta oto-laryng. (Supp. 10) **1**:156; (Supp. 11) **2**:1-213, 1929.

23. Johansen, H.: Relation of Audiograms to the Impedance Formula, Acta oto-laryng., Supp. 74, pp. 65-75, 1948.

24. Lüscher, E.: Experimentelle Trommelfellbelastungen und Luftleitungsaudiogramme mit allgemeinen Betrachtungen zur normalen und pathologischen Physiologie des Schalleitungsapparates, Arch. Ohren-, Nasen-, u. Kehlkopfh. **146**:373-401, 1939; Untersuchungen über die Beeinflussung der Hörfähigkeit durch Trommelfellbelastung, Acta oto-laryng. **27**:250-266, 1939; The Functional Effect of Loading of the Tympanic Membrane, ibid. **33**:265-272, 1945.

25. Veckmans, J. I.: Essai d'explication du recruitment, Acta oto-rhino-laryng. belg. **3**:355-360, 1949.

bone-conduction pathways (in the same ear) should show recruitment for bone conduction because of an increase in the mass component of impedance. For another example, the rise in impedance because of an increase in the friction component of impedance (such as found in Ménière's syndrome and "probably attributable to cochlear hydropsy") should give rise to recruitment.

B. Analogue of Recruitment as a Fatigue Effect: A normal-hearing ear may be partially deafened by a high-intensity sound, and equal-loudness matches run between that ear and the other, protected, ear. It is generally agreed that studies on stimulation deafness show the effect here to be centered in the hair cells. In such cases, recruitment appears, as Davis and associates noted. Unfortunately, their data were too scanty and irregular on this particular point to specify the type

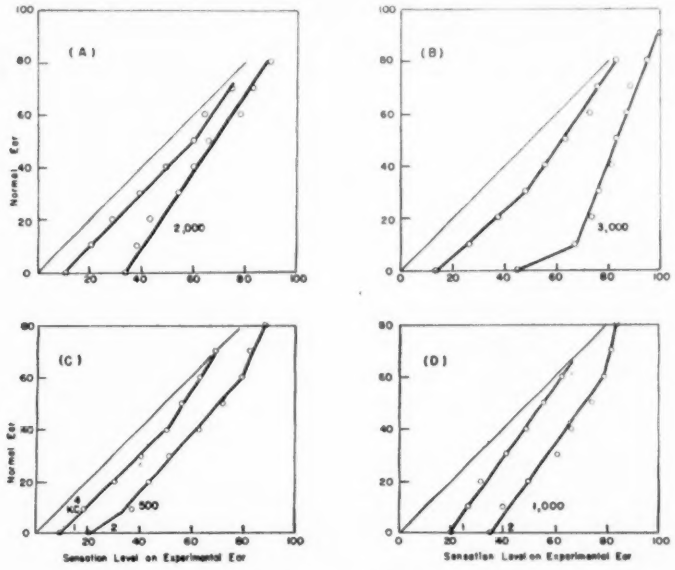

Fig. 16.—Analogue of recruitment in simulated deafness from fatigue.

of recruitment encountered. On a subject partially deafened on repeated occasions at a variety of frequencies and to a variety of degrees, we have found two types: straight-line and delayed recruitment. Figure 16 illustrates eight experimental sessions. It is clear that a fatigued ear always shows recruitment, probably completely, but that, especially for slight fatigue (of 20 db. or less), the delay may be rather great before recruitment starts. We have not seen such delays, of 40 and 50 db., in any of our patients. We have no explanation for this protracted delay with slight fatigue. We hesitate to suggest any one of several complicated hypotheses; but, until we obtain more experimental data, no simple explanation comes to mind.

C. Analogue of Recruitment as a Masking Effect: If a normal ear is masked by a noise, recruitment for a pure tone (or for speech) will occur in that ear, and

such recruitment is always of the asymptotic type. Furthermore, Steinberg and Gardner fully demonstrated that the constant loudness-loss explanation fits this case well.

A distinct difference is found, then, between fatigue and masking with respect to recruitment. The difference may simply be that fatigue affects the hair cells and thus simulates Ménière's disease in cases in which the nerve may be unaffected, while masking simulates nerve deafness by removing some fibers from action through the "line-busy" effect. Certainly, masking does introduce refractory periods in nerve fibers, but it is probably not correct to equate masking with nerve deafness. In true nerve deafness, for example, (1) recruitment often is extremely rapid, much more so than with a noise mask producing the same degree of threshold deafness, (2) loudness often reaches an early maximum, so that further increases in physical intensity produce no higher loudness, and (3) speech-articulation curves often reach an early maximum and then even decline, indicating only more garbling of speech with greater intensities. In masking by noise, on the other hand, the speech-articulation curve does not materially change its shape; its position on the intensity ordinate is merely shifted according to the mask intensity.

In masking, of course, other effects beside the refractory period are possible. There is the threshold shift, probably related to if not identical with the effects of residual fatigue, and there are the neural events of interaction, particularly inhibition. From the practical point of view, we conclude that the data from only those patients who exhibit asymptotic recruitment can be explained in terms of the neural mechanisms involved in masking.

D. Analogue of Recruitment in Bone Conduction: Veckmans states without citing evidence that a balance test between air and bone conduction reveals recruitment in the latter, since "the resistance due to bone conduction should be the normal resistance, plus a resistance due to mass." In the normal ear we have found it difficult to check this point because of cross conduction to the nonexperimental ear. We have had some success at relatively weak intensities by masking the unwanted ear, but we have conclusive results from a patient with one normal ear and the other ear stone-deaf from a skull fracture. At three frequencies no recruitment whatever was shown (Fig. 17 *B, C, D*). This confirms the results of our experiments with the use of masking on a subject with two normal ears (Fig. 17*A*) and seems to refute Veckmans' argument that high impedance at threshold (certainly the case with bone conduction) is always accompanied with recruitment.

It is, indeed, difficult to see how recruitment could occur for bone conduction as against air conduction—once the cochlea is entered, the two pathways are identical; the burden of explanation is rather on the side of those who would predict recruitment in this case.

6. *What Does Recruitment Loudness Contribute to Intelligibility?*—While the final curve has not been drawn relating the loudness of speech to the intelligibility of that speech, nevertheless, a great deal is known about the relation of speech intensity to intelligibility, and for the normal ear it is reasonable to assume that loudness itself makes a direct contribution from 0 to 100% intelligibility. For certain types of deafness, however, it may be that in certain regions an increase

in loudness will no longer improve intelligibility.[26] What is suggested by such cases is that the cues which go to make up good intelligibility are distorted by greater and greater intensities.

In our experiments, a number of patients with various types of disorder were studied further with intelligibility tests in the following way: (1) a curve of equal loudness for speech was determined for each patient; (2) at a selection of equally loud sensation levels, each patient received an intelligibility test in each ear,[27] and (3) a plot was drawn of points, showing per cent intelligibility in the two ears at equal loudness. Figure 18 illustrates this procedure, showing data for cases of conductive and perceptive types of deafness.

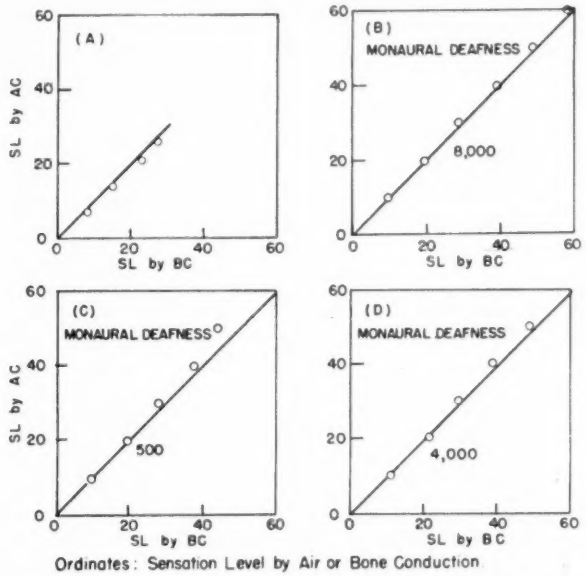

Fig. 17.—Graphs showing no analogue of recruitment for bone conduction versus air conduction. *SL* indicates sensation level; *BC*, bone conduction, and *AC*, air conduction.

It is clear that for a subject with two normal ears points derived as in Figure 18 would produce a 45-degree diagonal. Any systematic deviation from this line would indicate a disturbance of the normal relation between loudness and intelligibility. From the data in Figure 18 it can be seen that conductive deafness does not disturb this relationship but that a systematic deviation occurs for a patient with perceptive deafness. In this patient, the better ear yielded an intelligibility

26. Davis, H.: The Articulation Area and the Social Adequacy Index for Hearing, Laryngoscope **58**:761-778, 1948. Davis, H.; Morgan, C. T.; Hawkins, J. E., Jr.; Galambos, R., and Smith, F. W.: Temporary Deafness Following Exposure to Loud Tones and Noise, Acta oto-laryng., Supp. 88, pp. 1-57, 1950.

27. The test used consisted of several 50-word Harvard PB Lists, rerecorded in this Laboratory for constant intelligibility as recommended by Harris (Harris, J. D.: Some Suggestions for Speech Reception Testing, Arch. Otolaryng. **50**:388-405 [Oct.] 1949).

score of 50%, while the worse ear yielded 22%, even though the patient stated that the two intensities seemed equally loud. Again, at an equally loud intensity at which the better ear yielded 100%, the worse ear yielded only 68%, and the worse ear yielded no higher score when the loudness was further increased.

Thus, although it does not seem possible to agree with Steinberg and Gardner [28] that the audiogram alone is sufficient for prediction of speech-reception loss irrespective of the type of hearing loss being considered, nevertheless we conclude tentatively (in agreement with the findings in their one patient with nerve deafness for whom equal-loudness and articulation data are presented) that for some ears it is not possible to predict intelligibility from loudness measurements alone.

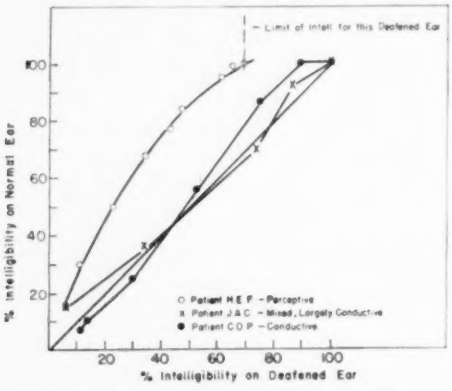

Fig. 18.—Comparison of intelligibility for speech, at equal loudness, for different types of deafness. Each point represents two intelligibility tests, one on each ear; for every point, speech sounded equally loud in the two ears (as determined by previous loudness matching).

SUMMARY AND CONCLUSIONS

The technique of alternate binaural loudness matching was used to explore the growth of loudness in the worse ear of patients with one defective and one normal ear. Data for speech and for pure tones were collected on a variety of types of deafness. It was desired to know whether an ear which exhibits recruitment for pure tones (i. e., an abnormally rapid growth of loudness) would also exhibit it for speech and, if so, with what characteristics. It was found that patients can be readily induced to make loudness matches for speech in two ears of unequal ability and that these matches can be as precise as those for steady pure tones. It was further found that irrespective of the type of deafness recruitment for speech followed a course parallel to that for pure tones and roughly intermediate among the curves of recruitment for pure tones in the speech range.

Four types of recruitment curves were distinguished: asymptotic, straight-line, delayed, and delayed plus asymptotic. No perfect correspondence exists between any type of curve and any type of hearing defect.

The question is examined whether recruitment is to be thought of as arising from a constant loss of loudness. Our data do not unequivocally support this view.

28. Steinberg, J. C., and Gardner, M. B.: On the Auditory Significance of the Term Hearing Loss, J. Acous. Soc. Am. **11**:270-277, 1940.

Rather, it seems that the course of recruitment curves often indicates a constant decrease in the decibel difference between the normal and the experimental ear.

It is thought that the delay feature of some recruitment curves may arise in any one of three ways: 1. The so-called normal ear may itself have recruitment to some extent, so that loudness balances show no differential rate in the two ears. 2. The deafness may have a conductive component. 3. The ear with recruitment may have a type of disability which affects threshold and so produces partial deafness, but this disability may be such as to be overcome more or less completely with intensities somewhat higher than of threshold strength.

A number of analogues of recruitment may be found in the normal ear by studying the effects of occlusion, of low vs. high frequencies, of fatigue, and of masking. These results, taken together with the different types of recruitment found in certain types of deafness, point to recruitment as quite possibly arising from any one of several sources. Certainly recruitment for fatigue is much different from that for masking, for example.

Some crucial results of experiments with bone conduction vs. air conduction weigh against Veckmans' explanation in terms of impedance. Lorente de Nó's explanation in terms of neural summation and occlusion, though it does not of itself explain varying rates and types of recruitment, remains a firm starting point. Quite possibly, varieties and rates of recruitment are related to whether all hair cells or nerve fibers are partially damaged, whether some are partially damaged and some destroyed, whether some are normal and some partially damaged, etc.

An indication is given that increasing the loudness of speech does not, for some patients with perceptive deafness, cause a proportionate increase in speech intelligibility.

STRUCTURE AND FUNCTION OF THE COCHLEAR AQUEDUCT

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THE COMMONLY accepted view about the cochlear aqueduct is that it provides an open connection between the perilymphatic space of the cochlea and the subarachnoid space of the brain. It is often said also that perilymph is simply cerebrospinal fluid. Yet there is clinical evidence that is difficult to reconcile with such views. It has long been known that in persons suffering from cerebrospinal meningitis the inner ear only rarely becomes involved and most often such persons pass through the terminal stages of this disease and finally succumb without giving any sign of auditory impairment. A postmortem examination then will show that the cerebrospinal fluid is teeming with pus cells, yet the perilymph is entirely free of them. It is puzzling how such a limitation of the infection can exist if there is an open communication of fluid along the cochlear aqueduct. This consideration has led us to examine further the much-debated question of the patency and function of the cochlear aqueduct.

During its long history this question has been approached in several ways.

1. STRUCTURAL INVESTIGATIONS

The first approach is naturally structural, and it dates from the discovery of the early anatomists that a canal passes through the petrous bone from the brain region to the tympanic scala of the cochlea. The discovery of this canal has been credited to Du Verney, though perhaps improperly. In 1683 he described a narrow passage running from a point below the internal auditory meatus to the scala tympani in the region of the round window and transmitting (he thought) an artery and a vein to supply the cochlea and other parts of the labyrinth.[1] The fact is, however, as Cotugno later made clear,[2] that in this region are two canals running side by side. One of them passes a vein and doubtless is the one that Du Verney saw, whereas the other contains a clear fluid that Cotugno regarded as labyrinthine fluid. The fluid lies inside a membranous lining of the bony canal that Cotugno believed to be an outgrowth of the dura mater.

From the Lempert Institute of Otology and the Lempert Research Foundation, Inc.

1. Du Verney, J. G.: Traité de l'organe de l'ouïe, Paris, 1863, p. 40.

2. Cotugno, D.: De aquaeductibus auris humanae internae, Vienna, 1774.

Cotugno regarded the aqueduct as a pathway for the flow of cochlear fluid inward to the brain. Pacchioni, on the contrary, had said that the cerebrospinal fluid flows outward to the ear.[3]

Many studies followed, in man and other animals,[4] and as histological techniques were perfected their results gave final verification of Cotugno's description of the cochlear aqueduct as separate from the venous pathway that came to be known as Cotugno's canal.[5] It became clear also that there are large variations in the course and size of the aqueduct in different animals and that in man and the other primates it is particularly long and narrow. There is no doubt about the patency of the duct in the lower mammals, but in the primates this fact is difficult to establish by microscopical examination.

The studies of primate ears show that at some places in the course of the duct, and especially near its junction with the scala tympani, the membranous lining almost fills the whole space. Some observers go further and assert that in this region no lumen is present. They therefore regard the aqueduct as functional in the lower forms but as only vestigial in man and the other primates. Thus Altmann and Waltner [6] in 1947 spoke of a loose, membrane-like condensation of tissue in the cochlear aqueduct of man near its opening at the scala tympani but noted that this tissue failed to form a continuous membrane. Then Waltner,[7] in a following article, spoke of this tissue as constituting a barrier membrane across the lumen and exhibited several sectional views in which the obstruction seemed complete. A number of writers have accepted this formulation and have come to refer to Waltner's "membrane" as a positive obstruction.

Our own observations have been made on 10 monkey ears in serial section and have included detailed measurements on one of them. The cochlear aqueduct is, indeed, long and narrow, as previous reports have shown. There are individual differences among ears, but typically the form is that of a flat S, with the greater curvature near the cochlear end. This form is shown in our sketch of Figure 1. From its widely flaring cranial end the passage narrows quickly and then takes a course that is fairly straight or sometimes gently curved, and then as the cochlea is approached it swings in a gentle arc to its opening in the scala tympani. There is often at this end also a great thickening of the bony wall adjacent to the round window, around which the aqueduct makes an abrupt turn, as shown in Figure 2.

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3. Pacchioni: De dura meninge humana, 1721, cited by Karbowski, B.: Vergleichende anatomische Studien über den Aquaeductus cochleae und über seine Beziehungen zum Subarachnoidalraum des Gehirns, Monatsschr. Ohrenh. **64**:687-715, 1930.

4. For reviews of these studies, see (*a*) Grünberg, K.: Zur Frage der Existenz eines offenen Ductus perilymphaticus, Ztschr. Hals-, Nasen-, u. Ohrenh. **2**:146-151, 1922. (*b*) Gerlach, H.: Über die Durchgängigkeit des Aquaeductus cochleae, Arch. Ohren-, Nasen- u. Kehlkopfh. **146**:17-22, 1939. (*c*) Karlefors, J.: Die Hirnhauträume des Kleinhirns, die Verbindungen des 4. Ventrikels mit den Subarachnoidalräumen und der Aquaeductus cochleae bei Kindern und Erwachsenen, Acta oto-laryng. (Supp.) **4**:1-184, 1924. (See especially the last article.)

5. It might more appropriately be called Du Verney's canal.

6. Altmann, F., and Waltner, J. G.: The Circulation of the Labyrinthine Fluids: Experimental Investigation in Rabbits, Ann. Otol. Rhin. & Laryng. **56**:684-708, 1947.

7. Waltner, J. G.: Barrier Membrane of the Cochlear Aqueduct: Histologic Studies on Patency of Cochlear Aqueduct, Arch. Otolaryng. **47**:656-669 (May) 1948.

Figure 1 is carefully drawn to scale and shows a canal about 3.8 mm. in length. This canal is lined with a loose arachnoid type of tissue, which fills the major portion of the space and leaves only a minute lumen. This lumen is narrowest near the cranial end, and at one place, as shown, is only 4.3 μ wide. (This size is smaller than that of the monkey's red blood corpuscles.) Though small in caliber, the lumen is continuous in all our specimens. There is no evidence of a "barrier membrane" as described by Waltner.

The cochlear aqueduct in man is generally similar in form and size to that just described for the monkey. Its lumen is likewise small; we have found it in places to be as small as 3.2 sq. μ in cross section.

2. PRESSURE RELATIONS

A second approach to our problem is in terms of pressure relations. Schwalbe [8] first pointed out that if there is a direct connection between the cerebrospinal fluid

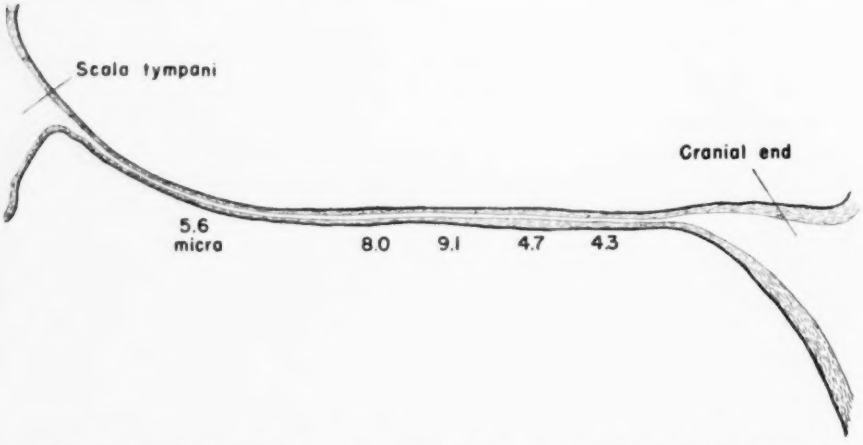

Fig. 1.—Scale drawing of the cochlear aqueduct in the monkey. The size of the lumen has been exaggerated twofold: horizontal scale, 1 mm.=20 μ; vertical scale, 1 mm.=10 μ. The numbers on the drawing show the width of the duct in micra at the five positions indicated.

and the perilymph by way of the cochlear aqueduct there should be a close correspondence between the pressures of the two fluids. On this basis Bárány [9] favored the use of lumbar puncture as a means of treatment for reducing a pathologically elevated labyrinthine pressure. Rejtö,[10] however, failed to observe any reduction of pressure in several ears treated in this manner and therefore came to doubt the existence of any open, direct connection through the aqueduct. He accepted only an indirect connection by way of blood vessels that he considered to run through the aqueduct.

8. Schwalbe, G.: Der Arachnoidalraum, ein Lymphraum und sein Zusammenhang mit dem Perichoroidalraum, Centralbl. med. Wissensch. **7**:465-467, 1869.

9. Bárány, R.: Der Bárányesche Symptomenkomplex, Diagnose und Therapie, Ätiologie und Prognose, Verhandl. deutsch. otol. Gesellsch. **21**:108-132, 1912.

10. Rejtö, A.: Über das Flüssigkeitssystem des Labyrinthes, Monatsschr. Ohrenh. **55**:324-330, 1921.

Kobrak, Lindsay, and Perlman [11] demonstrated in rabbits that pressure can be communicated from the cranial cavity to the labyrinthine fluid. They introduced a slowly alternating pressure through an opening in the skull and recorded the changes in a manometer introduced into the cochlea. These pressure changes may have been transmitted through the cochlear aqueduct, or alternatively through nerve foramina or blood vessels. It ought to be pointed out that the lumen of the cochlear aqueduct in the rabbit is considerably larger than in the monkey or man.

3. COMPARISON OF FLUIDS

A third approach to our problem is by a comparison of the physical and chemical properties of perilymph and cerebrospinal fluid. If the perilymph is simply cere-

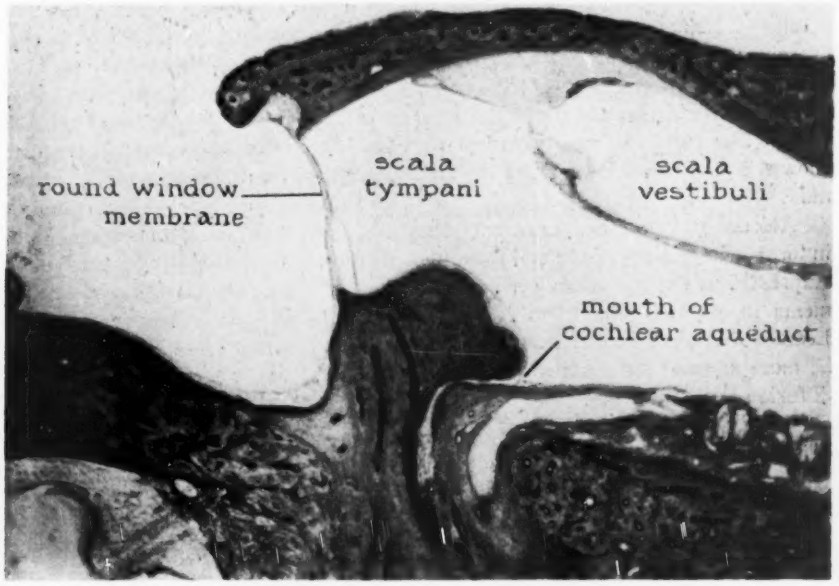

Fig. 2.—The cochlear aqueduct at its point of entrance into the scala tympani. Monkey 96-13, right.

brospinal fluid that enters the cochlea through the aqueduct, this fact should be revealed in similarities of the two fluids. A number of physical and chemical comparisons have been made.

Szász,[12] working with dogs, obtained a sample of labyrinthine fluid by pushing a capillary tube through the round window membrane, and obtained cerebrospinal fluid by cisternal puncture. He then compared the two fluids in terms of their refractive indices, which he reported as 1.335147 for labyrinthine fluid (mean of 17

11. Kobrak, H. G.; Lindsay, J. R., and Perlman, H. B.: The Next Step in Auditory Research, Arch. Otolaryng. **31**:467-477 (March) 1940.

12. Szász, T.: Beiträge zur Labyrinthliquorfrage, Ztschr. Hals-, Nasen- u. Ohrenh. **6**:256-260, 1923.

tests) and 1.335270 for cerebrospinal fluid (mean of 15 tests). From this evidence he concluded that the fluids were different and that the labyrinthine fluid, which he believed was mainly perilymph, was the less dense. Hence he concluded in favor of the view that perilymph flows inward through the cochlear aqueduct to the more concentrated cerebrospinal fluid. However, a statistical treatment of his data shows that the difference between the two fluids is not reliable. The probability is 30% that this difference would appear by chance alone in two sets of measurements on the same fluid, and so from this evidence we are not justified in concluding that the two fluids are distinct.

Ledoux [13] also measured the refractive indices of these fluids, but with different results. For the dog he obtained a value of 1.3349 for perilymph and 1.3342 for cerebrospinal fluid. The difference is in a direction contrary to the one reported by Szász but again is too small to be regarded as significant. In the cat Ledoux obtained a value of 1.33495 for perilymph (mean of 8 measurements) and 1.33427 for cerebrospinal fluid (mean of four measurements). There was no overlap of the readings, but the number of readings is too small for certainty as to their significance. This investigator further noted that after a sample of perilymph was taken the fluid was soon replaced and that measurements made on successive samples from a given ear showed a progressive lowering of the index until it approached that of cerebrospinal fluid.

Aldred, Hallpike, and Ledoux [14] compared perilymph and cerebrospinal fluid in terms of osmotic pressure and found this pressure to be the greater for perilymph. Expressed as the equivalent per cent of sodium chloride, the average of measurements in six cats was 1.046 for perilymph and 1.017 for cerebrospinal fluid. Ledoux [15] obtained similar results by a thermoelectric method that he considered to be more suitable for small samples. On the other hand, when he used a microdiffusion method for measuring the chloride content of these two fluids he failed to obtain any significant differences. His results for seven cats, expressed as per cent of sodium chloride, were 8.91 for perilymph and 8.83 for cerebrospinal fluid.

Another way of comparing these fluids is in terms of hydrogen-ion concentration. Ledoux [16] obtained a pH of 7.87 for perilymph and 7.45 for cerebrospinal fluid (each figure the mean for 11 cats). Kaieda [17] had previously made these measurements on fluids for the shark and had obtained a pH of 7.41 for perilymph and 7.23 for cerebrospinal fluid. The two sets of measurements agree in indicating a slightly greater alkalinity for perilymph.

Waltner and Raymond [18] obtained samples of human perilymph from the lateral semicircular canal during operations for Ménière's disease. They made comparisons

13. Ledoux, A.: Indice de réfraction des liquides céphalo-rachidiennes et labyrinthiques du chat, Acta biol. Belg. **1**:506-508, 1941.

14. Aldred, P.; Hallpike, C. S., and Ledoux, A.: Observations on the Osmotic Pressure of the Endolymph, J. Physiol. **98**:446-453, 1940.

15. Ledoux, A.: Concentration moléculaire totale des liquides céphalo-rachidiennes et labyrinthiques du chat, Acta biol. Belg. **1**:504-506, 1941.

16. Ledoux, A.: Le pH des liquides labyrinthiques (chat), Bull. Soc. roy. sc. Liège **12**:254-256, 1943.

17. Kaieda, J.: Biochemische Untersuchungen des Labyrinthwassers und der Cerebrospinalflüssigkeit der Haifische, Ztschr. physiol. Chem. **188**:193-202, 1930.

18. Waltner, J. G., and Raymond, S.: On the Chemical Composition of the Human Perilymph and Endolymph, Laryngoscope **60**:912-918, 1950.

with cerebrospinal fluid as to ultraviolet light absorption and protein content. They reported that there were marked differences between the fluids even when allowance was made for the fact that the perilymph sample was contaminated with blood.

This problem of the contamination of fluids has had far less consideration than it deserves. Actually it is not possible to take any sample of perilymph or of cerebrospinal fluid without contamination with blood and injured tissues. The matter is only moderately serious for cerebrospinal fluid because the fluid is plentiful and the foreign material is small in comparison, but for perilymph it is very serious indeed. Waltner and Raymond believed that they could allow for the contamination by noting the extent to which their samples absorbed those wave lengths of ultraviolet light that are characteristically absorbed by blood, but such a procedure is only crude at best. It has to be assumed that the composition of the contaminating blood is the same as that of whole blood, which is not true for the exudate of fine capillaries. The actual contamination thus may be much greater than this method indicates. In our experiments we were unable, in spite of all precautions, to obtain a sample of perilymph through the round window without appreciable blood contamination. Microscopical examination of the sample always revealed blood corpuscles. Also, if a fluorescent dye (sodium fluorescein) had previously been injected into the blood stream, the sample was always fluorescent under ultraviolet light. That this fluorescence is not due to the secretory diffusion of the dye into the perilymph, but is a contamination that occurs as the sample is taken, is shown by the fact that it is of the same order of magnitude regardless of whether the sample is taken 5 minutes or 20 minutes after the injection.

4. EFFECT OF OCCLUSION OF THE AQUEDUCT

If a patent aqueduct is a functional necessity, then its occlusion should lead to functional disturbances. If, for example, perilymph is derived from cerebrospinal fluid and if it is continually being used up and being replaced by a flow through the aqueduct, then a closure of this pathway should lead to an obvious deficiency.

Karlefors [19] in 1924 reported cases in which hearing was present before death and subsequent histological study showed places in the course of the cochlear aqueduct where the lumen seemed to be occluded.

Uyama [20] produced a blocking of the cochlear aqueduct of rabbits by two methods. One method was to inject an emulsion of sarcoma material into the cisterna magna, which in some cases produced a growth at the cranial entrance of the aqueduct and within its lumen and caused a partial or complete occlusion of the lumen. Another method was to expose the cranial space and to insert a fine thread of catgut into the lumen. The plug of catgut was augmented by the foreign-body reaction of the tissues and effectively blocked the lumen. The blockings were checked by histological examinations. The effects on the cochlea and other parts of the labyrinth were studied several weeks after the blocking, and it was found that Reissner's membrane was bulged into the scala vestibuli in varying degrees, depending on the thoroughness of the blocking and its duration. Sometimes this bulging was so extensive as practically to eliminate the scala vestibuli. In spite of these changes, the organ of Corti was everywhere present and of good appearance.

19. Karlefors.[4c]

20. Uyama, Y.: Histopathologische Veränderungen im Innenohre, bedingt durch den experimentellen Verschluss des Aquaeductus cochleae, Okayama-Igakkai-Zasshi **45**:1128-1150, 1933.

Uyama concluded from these observations that the perilymph comes from cerebrospinal fluid and that its principal path of entrance is through the cochlear aqueduct. The reasoning is that the blocking of the cochlear aqueduct has cut off the supply of perilymph and absorption of this fluid within the cochlea has produced a deficiency and that the available space is now taken up by the endolymph by bulging out Reissner's membrane.

It needs to be pointed out that Uyama's conclusion does not necessarily follow from his observations. An increased secretion of endolymph causing a bulging of Reissner's membrane might be a direct consequence of a diseased condition of the cochlea. This explanation implies that the infection or the foreign-body reaction was transmitted from the aqueduct to the cochlea, but it does not lead to Uyama's conclusion that the source of perilymph is the cerebrospinal fluid.

We must further point out that, whether or not this interpretation of Uyama's is accepted, the good condition of the organ of Corti is surprising. Either tissue reactions or pressure changes might be expected to cause degenerative changes of this organ. Still, taken at their face value, the results indicate that the patency of the aqueduct is not essential to the persistence of the sensory structures.

5. INJECTION METHODS

The commonest method of investigating the patency of the cochlear aqueduct is the injection into the cochlea or into the subarachnoid space of fluids that have been made identifiable by the addition of dyes or particulate matter and a later microscopical examination to discover the distribution of the foreign material in the tissues. Cotugno seems to have been the originator of this technique when he proved the patency of the bony aqueduct by forcing mercury through it.

The modern use of this method began with Schwalbe,[21] who attempted to inject a suspension of ferric ferrocyanide (Prussian blue) into the subdural space of rabbits. He reported that the particles entered the labyrinth through the cribriform plate along the openings that carry the twigs of the cochlear nerve. Others, beginning with Weber-Liel,[22] promptly repeated Schwalbe's experiment and found generally that the particles of pigment passed to the cochlea along the cochlear aqueduct and sometimes also entered a little way along the perineural route as Schwalbe had described. It also became clear that Schwalbe had damaged the arachnoid membrane and had introduced the pigment into the subarachnoid as well as into the subdural space. It is the subarachnoid space that communicates with the cochlear aqueduct, as the results for the lower animals clearly show.[23]

Less extensive study by this method has been made on the primates. Altmann and Waltner in their recent investigation included a series of monkeys. They used Weed's method, injecting a mixture of iron ammonium citrate and potassium fer-

21. Schwalbe.[8]

22. Weber-Liel: Experimenteller Nachweis einer freien Communication der endolymphatischen und perilymphatischen Räume des menschlichen Ohrlabyrinthes mit extra-labyrinthischen intracraniellen Räumen, Arch. path. Anat. **77**:207-226, 1879.

23. See the recent work of Jampolsky, L. N.: Über den morphologischen Zusammenhang des Subarachnoidealraums mit dem Labyrinth, Monatsschr. Ohrenh. **69**:23-34, 1935. Altmann, F., and Waltner, J. G.: The Circulation of the Labyrinthine Fluids: Experimental Investigations in Rabbits, Ann. Otol. Rhin. & Laryng. **56**:684-708, 1947; New Investigations on the Physiology of the Labyrinthine Fluids, Laryngoscope **60**:727-739, 1950.

rocyanide solutions into the cisterna magna. Later, after the animal was killed, a treatment of the tissues with hydrochloric acid caused a precipitation of particles of ferric ferrocyanide. They found these particles in the cranial portion of the cochlear aqueduct, and in one of nine monkeys in the cochlea itself. These observations are in contrast to the ones that they made on rabbits and cats, in which the particles were found generally in the scala tympani and often in the scala vestibuli as well.

Our own injection experiments were carried out in seven monkeys. We used an aqueous suspension of colloidal carbon [24] for injection into the cisterna magna.

EXPERIMENTS 1 AND 2.—The first two experiments were of a preliminary nature. The carbon suspension was injected into the cisterna and then a gross examination was made to ascertain whether the particles could be found in the perilymph. In the first experiment the round window was opened 20 minutes after the injection, and no carbon was seen. In the second experiment the lateral semicircular canal was opened 5⅓ hours after the injection and 20 minutes later the round window was opened and a large part of the cochlear wall was removed, all without revealing any trace of the injected material. In the remaining animals the examination was made after histological preparation of the tissues.

In three experiments 1 cc. of cerebrospinal fluid was removed through a spinal needle, and then this volume of the carbon suspension was immediately introduced. Summaries of the observations follow.

EXPERIMENT 3.—The animal was killed 30 minutes after the carbon injection by a perfusion of fixing fluid through the carotid artery. Histological examination showed carbon particles in the cranial mouth of the cochlear aqueduct on both right and left sides but none beyond the middle portion of the aqueducts. A small amount of carbon was seen about the eighth nerve, but none had passed through the cribriform plate.

EXPERIMENT 4.—This animal was killed by perfusion seven days after injection of carbon particles. On examination, no particles were found in the cochlear aqueduct on the right side. The histological preparation failed to include the cranial mouth of the left aqueduct, but its remaining portion was free of particles. As before, a few particles were seen around the eighth nerve but none had passed through the cribriform plate.

EXPERIMENT 5.—In this animal the stapes was removed on the right side and the injection of carbon was continued at a pressure of 12 in. (30.5 cm.) of water for 1¼ hours. The histological study showed carbon particles in the right aqueduct all the way from its cranial mouth to its middle portion. No particles were found in the aqueduct on the left side.

In the next two experiments the injection was sustained under moderate pressure for a considerable time.

EXPERIMENT 6.—The round-window membrane was destroyed on the left side, and the injection was continued under a pressure of 8 in. (20.3 cm.) of water for 2⅓ hours. The right ear showed a considerable amount of carbon in the mouth of the aqueduct and some in its midportion.

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24. The material, known commercially as "Aquadag," was supplied by Mr. A. Crankshaw of the Acheson Colloids Corporation.

The left ear of this animal showed the most extensive penetration of carbon particles of all our specimens. The aqueduct contained a dense mass of particles at its cranial mouth and progressively smaller amounts along its course (Fig. 3). A few particles were found at the cochlear end, embedded in the loose tissue there, but none had penetrated into the cochlea itself. Several masses of particles were seen about the eighth nerve on both sides. They were crowded close to the cribriform plate and a few had penetrated its outermost openings. None, however, had gone all the way through this plate. The reason, as Figure 4 clearly shows, is that the nerve bundles fully fill their foramina.

EXPERIMENT 7.—In this experiment the stapes was removed on the right side and the injection was continued at a pressure of 12 in. (30.48 cm.) of water for

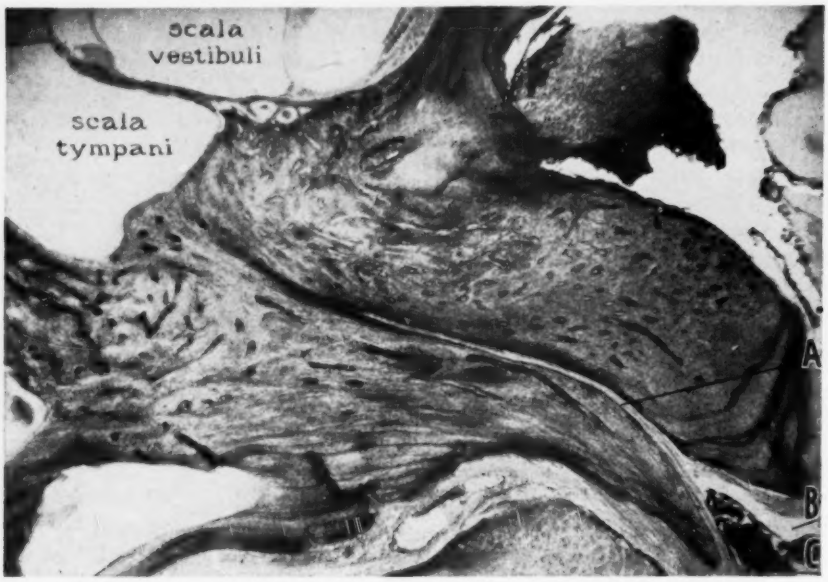

Fig. 3.—The cochlear aqueduct is shown in this section for about three-fourths of its course; *A*, cochlear aqueduct; *B*, cranial mouth; and *C*, mass of carbon particles. Carbon particles are present in quantity at the cranial mouth at the lower left of the figure, and some have penetrated about halfway to the cochlea. No particles were found in the scala tympani. Monkey 98-14, left.

two hours (Fig. 5). Examination showed on the right side a plentiful amount of carbon at the cranial mouth of the aqueduct and then a very rapid thinning out (Fig. 6). None was found at the cochlear end or in the cochlea itself. The left side showed a similar condition except that the particles did not penetrate as far along the aqueduct.

It is evident in these observations that when the cochlea is intact, as in the first five experiments, the cochlear aqueduct is penetrated only with great difficulty if at all. The reason lies in two conditions, the small size of the lumen and the fact that the cochlea is already filled. Operating here is a simple hydraulic principle: It is not possible to introduce fluid into an already filled container when there is

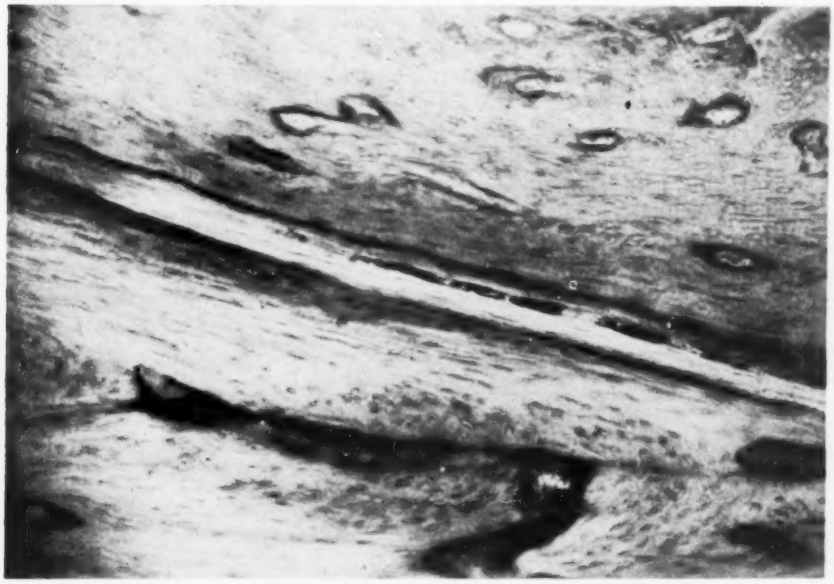

Fig. 4.—High-power view of the middle portion of the cochlear aqueduct of Figure 2. Note the masses of carbon particles which fill the lumen of the canal at certain places.

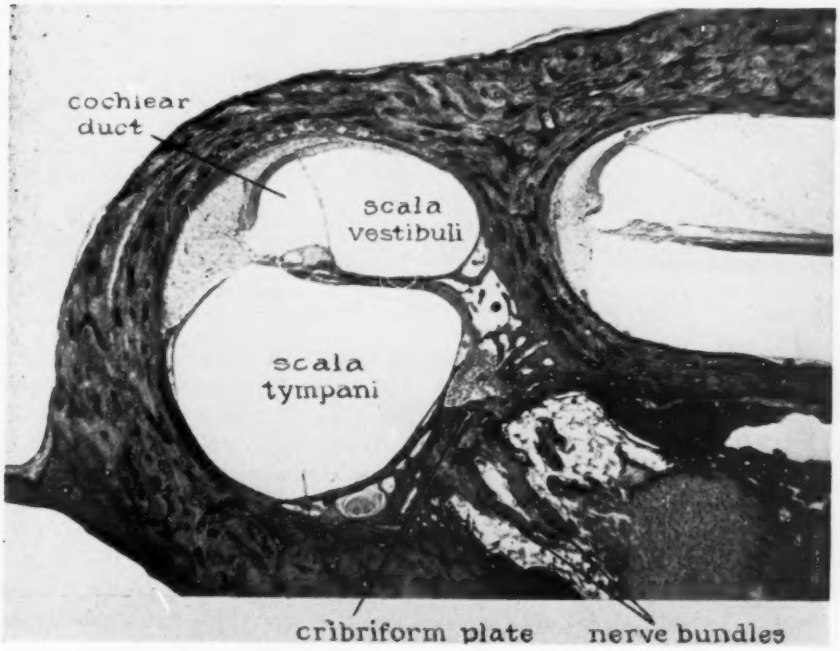

Fig. 5.—Large masses of carbon particles are seen in the spaces about the cochlear nerve, and on the left some particles are seen around the nerve bundles at the cribriform plate. Monkey 100-16, right.

no means of egress. When this means is provided, as in the last two experiments, the penetration of the aqueduct is made easier. It thus turns out to be unnecessary to assume the presence of a barrier membrane in the aqueduct, as Waltner did, to prevent a free flow into (or out of) the cochlea. Under normal conditions there might be a diffusion of fluid along the duct, which would be exceedingly slow, but there can be a true flow of fluid through it only if there is differential secretion and absorption at its two ends—only if there is an excess of production of perilymph in the cochlea and a disposal of fluid somewhere in the cerebrospinal system or, contrariwise, an absorption of perilymph in the cochlea and an excess of production in the cerebrospinal system. If, on the other hand, there is a fluid equilibrium within each of these two systems—if both the labyrinthine system and the

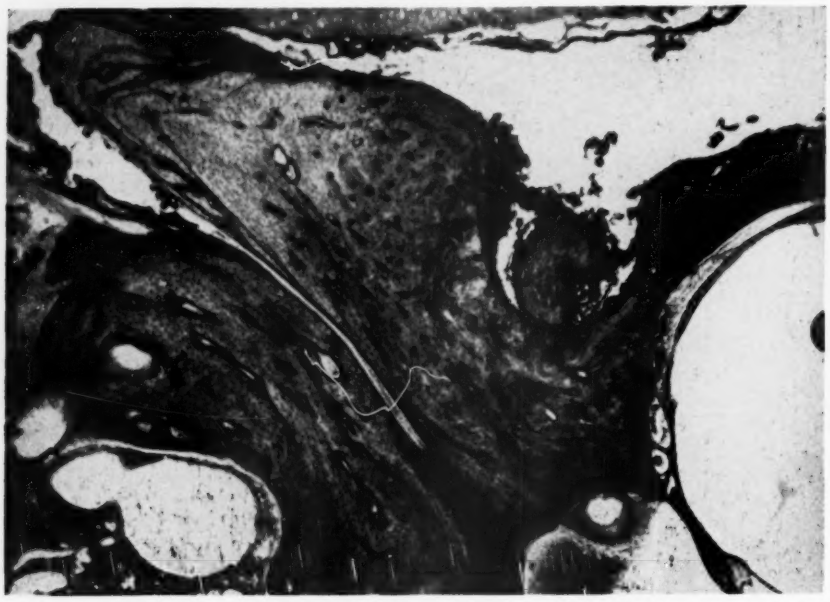

Fig. 8.—The cranial mouth of the cochlear aqueduct shows large clumps of carbon particles, but none had penetrated far into the aqueduct proper. Monkey 100-16, right.

cerebrospinal system maintain their fluid contents by a balance of secretory and absorptive processes—then there is no need for a continuing flow through the aqueduct in one direction or the other. This is the most likely state of things.

This view of the problem of fluid flow through the cochlear aqueduct is consistent with the larger part of the evidence as reviewed above. The studies of structure indicate that in the lower mammals there is a fairly wide lumen through the aqueduct, and in the primates there is a continuous lumen but only a very narrow one. There are indications that fluid pressure can be transferred through the aqueduct, but in the primates this transfer encounters very great frictional resistance. The physical and chemical comparison of perilymph and cerebrospinal fluid shows many similarities and only slight differences, but similarities do not necessarily signify that the two fluids are secreted in a common place. They are

equally well explained as products of secretion in different places by similar tissues. The results of the experiments on occlusion of the aqueduct in rabbits indicate that a communication normally exists by this pathway in this species. Yet it is of interest that a blocking of this pathway for a period of weeks has no very serious effect upon the organ of Corti.

Our conclusion is that the cochlear aqueduct is normally patent in all forms but that there is no evidence of active flow through it. Indeed, in the primates such a flow is prevented by the minute size and consequently great resistance of the lumen. If there is no active flow through the aqueduct, we still have the question of the function of this structure. The answer may be that it has no specific function but is merely vestigial. For the primates beyond the embryonic stage this possibility seems especially likely.

If this view of the labyrinthine and cerebrospinal fluid systems is the true one, the clinical problem with which we began becomes clear. Infection does not readily pass along the cochlear aqueduct, because there is no systematic fluid flow. Infection can creep along this path, of course, as it can make its way anywhere in the body, but only at a comparatively slow pace and in the face of the body's combative resources. There is the further possibility that when infection is present the cells lining the aqueduct become edematous and reduce further the already narrow lumen or block it altogether. We can say much the same about the perineural route between the cranial cavity and the cochlea. This route also seems to impose a considerable barrier to the movement of fluid and especially of particulate matter, because the nerve bundles effectively plug the openings through which they pass. Hence we can explain why a meningitis can occur alone without the complication of labyrinthitis, and why a labyrinthitis can occur alone also, as clinical experience proves.

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THE ADENOID PROBLEM

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UNTIL recently the nasopharynx was the most neglected territory in the otolaryngologist's domain. Adenoidectomy is still the most unsatisfactory surgical procedure that he is called upon to perform. The majority still attempt to remove a highly vascular, loosely organized, and indefinitely demarcated structure from a cavity of irregular contour without the aid of sight. After use of the La Force adenotomes, the index finger is employed in an effort to detect remnants. These remnants are attacked either by friction with the gauze-covered finger or by curettes. The former method is largely useless. The latter not infrequently removes the full thickness of mucosa or cuts away cartilage from the tubal prominences, with resulting scarring. That these methods leave something to be desired, especially in children with conduction hearing problems, would seem well attested by the recent widespread popularity of the radium applicator. By some, this instrument has even been employed routinely after tonsillectomy and adenoidectomy.

It is the purpose of this paper to describe in detail a technique for performing adenoidectomy under direct vision in such a manner as to insure a macroscopically clean nasopharynx. It will be shown that the alleged inaccessibility of certain recesses of the nasopharynx, particularly Rosenmüller's fossae, is a myth, and that with proper methods the entire space is surgically approachable.

Attempts to achieve such a result are not new but date back to the beginning of this century. Proper appreciation of the modern technique is enhanced by a knowledge of these earlier attempts. Sidney Yankauer deserves credit for being the first in this country to interest himself in the nasopharynx. In the same year in which Edgar Holmes brought forth his nasopharyngoscope, Yankauer [1] published a paper containing an admirable description of the nasopharynx as seen under direct vision by means of his newly devised speculum. He had his patients sit upright and used no suction. Illumination was provided by an ordinary head mirror. Only one side at a time could be examined. Yankauer credited A. von Gyergyai with having made the boldest previous attempts at direct examination and surgery in the nasopharynx. Gyergyai,[2] a Hungarian, in 1910 presented a method of direct access to the nasopharynx by means of straight tubes of various lengths and thicknesses, illuminated by a Bruening electroscope. His patients lay

1. Yankauer, S.: The Pharyngeal Orifice of the Eustachian Tube, with a Description of a Speculum and Other Instruments for the Direct Examination and Treatment Thereof, Tr. Am. Laryng., Rhin. & Otol. Soc., 17th Annual Meeting, 1911, p. 178.

2. von Gyergyai, A.: Demonstration einer neuen direkten Methode zur Untersuchung und Behandlung der Pharyngealtubenmündung und des Innenraumes der Ohrtrompete, Versammlung der deutschen otologischen Gesellschaft in Dresden, May 13-14, 1910; abstracted in Zentralbl. Ohrenh. **3** (No. 10):449, 1910; cited by Yankauer.[1]

supine, head over the end of the table and throat cocainized. The soft palate was drawn forward with a finger and the tube placed. Constant suction was employed. Gyergyai was able to introduce a tubular speculum into the Eustachian tube as far as the isthmus. With this he could illuminate the middle ear so that another observer could view it. He was able to operate upon the sphenoid bone, cavernous sinuses, and pituitary body with this method. (He invented olives for dilating the Eustachian orifices, which are still used today.) Yankauer claimed for his own nasopharyngeal speculum a curvature more adaptable than the straight tubes of Gyergyai.

Joseph C. Beck [3] introduced the practice of retracting the soft palate by means of small rubber catheters placed through the nose. This method had merit, but was not completely satisfactory.

Isaac D. Kelley [4] invented a "direct vision adenotome" featuring a hood over the cutting mechanism, which served as a palate retractor, and through which one could obtain a very limited direct visualization.

It remained for Andrew Love to furnish the definitive method of direct access to the nasopharynx. In 1932 he evolved his soft palate retractor and an adenoid punch.[5] The retractor permitted for the first time a direct view of the entire nasopharynx, and its very simplicity was the more remarkable in view of the ponderous armamentarium which preceded it. As a piece of functional design it is a veritable work of art. Love stressed the inadequacy of the index finger for detecting adenoid remnants after the usual blind operation and mentioned the usefulness of the retractor for the clinical direct examination of the nasopharynx. For this he employed local anesthesia with butacaine (butyn®) sulfate or tetracaine (pontocaine®) hydrochloride. A number of progressive otolaryngologists began to use this instrument, but Love's contribution has never received the attention it deserves.

Louis Guggenheim [6] in 1941 described a procedure which he called "direct adenoidectomy." Using the Love retractor for exposure, he evolved a method of meticulous dissection under direct vision with a set of specially devised instruments for use after the La Force adenotomes. He was the first to emphasize the importance of meticulous adenoidectomy in cases of conduction hearing impairment or recurring otitis media and to report clinical results.[7] It was probably this feature of the work which began finally to attract some attention to the problem of the adenoid. Without doubt, it is no coincidence that after the publication of Guggenheim's paper the nasopharynx "came into its own." Previously neglected in the literature, it quickly became the subject of a veritable avalanche of publications. Interestingly, these publications, while deploring previous neglect of the subject and again stressing the inadequacy of the prevailing surgical adenoidectomy, ignored the proposed direct procedure and looked instead to irradiation as the solution of the problem. The radium applicator, used for some years in a very restricted manner, became suddenly and enormously popular. Being a technically

3. Beck, J.: Removal of Adenoids by Direct Inspection, Ann. Otol., Rhin. & Laryng. **22**:273 (June) 1913.

4. Kelley, I. D., Jr.: Direct Vision Adenotome, J. A. M. A. **79**:300 (July 22) 1922.

5. Love, A.: A New Soft Palate Retractor and Adenoid Punch, Arch. Otolaryng. **19**:66 (Jan.) 1934.

6. Guggenheim, L.: Direct Adenoidectomy, Arch. Otolaryng. **33**:314 (Feb.) 1941.

7. This instrument is made by V. Mueller and Company and the Storz Company.

simple procedure—and one, incidentally, for which large fees could be charged—its use has been by no means confined to otolaryngologists. It has been widely employed by general practitioners, pediatricians, and others. Is it not possible that had the eminently satisfactory procedure of direct adenoidectomy been adopted instead this all-important segment of otolaryngologic surgical practice would by now be generally recognized as beyond the capacity of anyone but a trained otolaryngologist?

It seems timely to redescribe this procedure and to emphasize again the steps which led to its development. Between 1936 and 1941, a large number of children were studied who had already had tonsillectomies and adenoidectomies performed by excellent men, yet direct visualization of their nasopharynges revealed significant remnants of adenoid tissue, especially in the lateral aspects of the vault.[7] Many of these patients had been operated on too recently for regrowth to have occurred. Examination of the nasopharynges of these children was first attempted with the smallest-sized mirror. It was found that only a few children could be successfully examined thus, but it was learned that the nasopharynx of the unanesthetized child could be directly examined in the supine position, head slightly over the edge of the table, with use of the Andy Love palate retractor and a tongue depressor. It was first explained to the child that the procedure would be uncomfortable but not actually painful, and that it would last only a moment. Very small children were simply mummied. The view of the whole field at a glance, though not as good as in the anesthetized patient, was adequate for diagnosis. Immediately after the examination, the child was brought to a sitting position and given a glass of water. This promptly terminated any crying. It was felt that this procedure was far more satisfactory to the examiner and considerably less alarming to the child than the use of the nasopharyngoscope. (To those unfamiliar with this method of examination, numerous attempts will be found necessary before satisfactory visualization and interpretation is attained with the single glance. Some experience at the operating table, where one can study the territory with more leisure and exposure, helps perfect technique. It should be emphasized that gentleness in retracting the palate is most important, even more so than at surgery, where the tissues are relaxed. Excessive force may even tear the soft palate. There should be no bleeding.) It was soon concluded that the problem presented by these children could best be solved by a refinement of the usual procedure of adenoidectomy.

TECHNIQUE OF DIRECT ADENOIDECTOMY

Anesthesia must be deep enough to abolish the pharyngeal reflexes and assure relaxation of the pharyngeal musculature. The practice of producing very light anesthesia for a "quick operation" will not permit adequate direct visualization of all parts of the nasopharynx (although in very young children such light anesthesia is admittedly safer).

After removal of the tonsils, the soft palate is elevated with the Andy Love retractor. First is seen the central adenoid mass and an accurate impression gained of its size (Fig. *A*). The retractor is withdrawn and, with the palate in its normal position, the largest possible La Force adenotome is introduced. Most of the central mass is removed with three or more bites. At this point it is well to control bleeding by means of tampons slightly moistened with epinephrine. After this, direct inspection with the Love retractor will almost always reveal further masses

which can be adequately removed under direct vision with the smallest La Force adenotome. (I use a specially constructed instrument, the basket of which measures only 9 mm. inside width.) This much of the total procedure should be routine in every tonsillectomy and adenoidectomy, irrespective of the indication. The last-noted step, if practiced faithfully after the usual blind finger examination and clean-up maneuvers, will convince the most skeptical of its value. Unfortunately, in many operating rooms proper equipment for the direct inspection of the nasopharynx is not in evidence. Many surgeons operate only with ceiling lights. This is not adequate for viewing the vault. One needs brilliant illumination from the operator's head. A survey of the usual tonsillectomy and adenoidectomy instrument tray, again, discloses no instrument for retracting the soft palate properly. Neither the retractor end of the Weder tongue depressor nor the Hurd retractor is adequate for viewing the vault. The Yankauer speculum gives a view which is really endoscopic and through it one can accomplish little but poking about in semidarkness with small forceps or punches. Compared with the use of the Andy Love retractor, the use of these methods is like watching a ball game through a knothole when one could easily look over the top of the fence and see all.

When the nasopharynx is as clean as it can be got with La Force adenotomes and bleeding is again controlled, the picture presented to the surgeon with direct visualization is new and unfamiliar—quite unlike anything he is used to in his experience either with mirror or nasopharyngoscope. A reorientation is necessary. The tubal prominences do not appear nearly so prominent as when seen by the other methods; indeed, they are often scarcely perceptible elevations. Rosenmüller's fossae, which look so inaccessible by other methods of examination, appear as shallow grooves stretching almost flatly before the operator. Viewed thus, they could hardly be more than a few millimeters in depth, measuring from the general level of concavity of the vault. This depth dimension differs, it is true, from a more commonly accepted measurement of "depth" of the fossae as conceived from the mirror or nasopharyngoscopic appearance—one which extends from the anterior lip of the torus to the lateralmost recess of the fossa. This measurement would appear in the direct view to be rather the length of the trough-like fossae than their true depth. (Both conceptions are undoubtedly valid from their own points of view, but the one herein advocated is basic to a consideration of the surgical accessibility of the fossa.) The Eustachian orifices appear as insignificant vertical slits or dimples.

It is interesting that these novel features of the direct view are not new but were quite well understood by Yankauer,[1] who gave an elaborate description of the geometrical optics of the situation which is worth quoting:

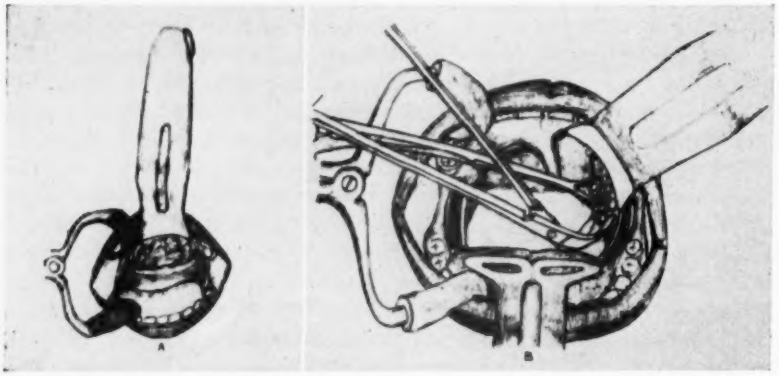
> The appearance of any organ when examined by the direct method differs from its appearance in a reflecting instrument, owing to the absence of inversion of the picture and to a better definition of the details of the surface. In addition to these there is another special peculiarity to the direct examination of the nasopharynx.
>
> When the nasopharynx is examined with a reflecting instrument, the mirror or reflecting prism is placed in the oropharynx. The line of vision is the same as if the eye were placed below and looking up into the cavity. The line of vision is therefore parallel to the posterior wall, oblique to the fornix, and perpendicular to the vault of the nasopharynx. Under these circumstances the curvature of the walls of the cavity is very evident to the eye; in fact, it is even exaggerated.
>
> On the other hand, when looking at the posterior wall through the direct speculum, the line of vision is perpendicular to the posterior wall; when looking at the vault, the patient's head is

thrown backward, and the line of vision is perpendicular to the vault. Each part of the nasopharyngeal wall therefore presents itself in exactly the same way as every other part, so that the entire wall looks like a continuous straight surface. The same is true of the posterior lip of the tube, so that in looking, e. g., at the orifice of the Eustachian tube, the eminence, which takes a horizontal direction above it, appears in the speculum as a vertical ridge behind it; the deep recess above the eminence appears as a vertical groove behind the ridge; and the vault appears as a vertical surface.

In view of the confusion over the "depth" of Rosenmüller's fossa, his description of this structure is interesting:

The fossa of Rosenmueller may therefore be described as a triangular space situated behind and above the cartilaginous portion of the tube, and corresponding in length with this cartilage, i. e., about one inch.

Some experience is required before the operator is "at home" and can identify the various structures with certainty, particularly the slit-like Eustachian orifices. One often sees on and around these structures collections of lymphoid tissue too small to be ablated with La Force adenotomes. One also sees the lateral curtains



(*A*) The soft palate is elevated with the Love retractor, exposing the central adenoid mass. (*B*) Love palate retractor obliquely placed with its lip hooked behind posterior tonsil pillar, exposing lateral curtain. Triangular holding forceps shown pulling curtain anteriorly and medially while Guggenheim scissors begins excision from below upward.

of lymphoid tissue along each lateral nasopharyngeal wall, extending onto the posterior surface of the corresponding posterior pillar. This relationship is impossible of demonstration without oblique retraction of the palate. It is easily observed that these curtains tend to fall over the Eustachian orifices with resulting obstruction. They give the impression of being distinct anatomic entities. L. K. Guggenheim [6] was the first to describe them and to point out the importance of removing them in cases of hearing difficulties or of otitis. This step cannot be accomplished with either adenotomes or curettes. Sharp dissection is the method of choice, although good results may be obtained with a punch forceps. This step requires strong lighting, an assistant, and skill in performing delicate dissection in a small enclosed space. It was found that ordinary tonsil scissors were too bulky and too straight for this dissection. A small right-angled curved scissors was therefore invented for this purpose.[7] The curtain is first seized with a small triangular holding forceps or with two Allis clamps "in series," while the soft palate is being

obliquely retracted by the operator. The clamps are then handed to the assistant who retracts medially and upward. The operator then insinuates the blades of the scissors about the lower end of the curtain, below the clamps, and dissects upward (Fig. *B*). This procedure begins in the oropharynx and ends high in the lateral wall of the nasopharynx. It is sometimes advisable to withdraw the scissors, replace the clamps, and begin afresh. When the curtain is finally hanging by a superior "pedicle," this pedicle can be severed or often simply avulsed. If this process is done carefully, no injury occurs to the posterior pillar or other important structures. When the curtain is small or excessively friable, it is sometimes sufficient to "milk" it with the triangular holding forceps. One avulses thus the lymphoid tissue present and leaves only a slightly redundant fold of mucous membrane. The small collections of lymphoid tissue remaining about the tubal prominences or in Rosenmüller's fossae can be similarly removed by dissection or avulsion. When these steps have been properly carried out, one has achieved a surgically clean nasopharynx. It is quite true, of course, that microscopic collections of lymphoid tissue remain because no adenoid capsule exists and because this tissue extends into the lining membrane which is not removed. There is no convincing evidence that such microscopic remnants are per se of pathologic significance.

In cases of hearing difficulties, after completion of the adenoidectomy proper, the Eustachian orifices are gently dilated, one at a time, with the Gyergyai olives, right and left. Gentle pressure is maintained for 25 seconds.

Follow-up testing of hearing is routinely done at the end of three months. The only complication encountered in my experience has been the very occasional production of an otitis media, which responds readily to penicillin. Myringotomy for effusion has been necessary in rare cases. The commonest cause of a few inadequate results has been an overlooked nasal condition—either allergy or chronic purulent maxillary sinusitis or both. Appropriate treatment of those conditions will rescue the patient unless they have already produced recurrence of an adenoid, in which instance further attention to the adenoid itself is also necessary.

RADIATION THERAPY

In a few cases there will be small, true postoperative recurrences of adenoid tissue, usually about the tubal orifices. Such instances are more likely in allergic children, and proper medical allergic management by the otolaryngologist tends to prevent them. Under such circumstances one may well hesitate to subject a child to another surgical procedure, although surgical management of such small recurrences is perfectly feasible. However, other methods seem justifiable at times. It is best first to try the treatment of Harold Lillie, of the Mayo Clinic. This consists of a course of strong iodine solution U. S. P. (Lugol's solution) by mouth, starting with 3 drops, three times a day, in water or milk with meals and increasing until some gastrointestinal reaction occurs (generally not more than 8 or 10 drops for children). One also uses nasal shrinkage and infrared heat applied to the ears plus pneumatic massage with the Seigle otoscope. If improvement is insufficient, it is preferable to send patients to a radiologist, who gives four x-ray treatments to both sides of the nasopharynx. The case is then restudied. Rarely, a second course of four treatments may be necessary. Details of the technique are as follows: At the first treatment 75 to 100 r is administered to each side of the nasopharynx

externally. The target-skin distance is 50 cm., voltage 220 kv., filtration 0.25 mm. Cu (half-value layer 0.85 mm. Cu). At the second treatment, if the child is cooperative, an intraoral port 3.5 to 4 cm. in diameter is used. The right and left sides are irradiated with 50 to 75 r each (140 kv., target-skin distance 35 to 50 cm., filtration 0.25 mm. Cu [half-value layer 0.42 mm. Cu]). Treatments are thus alternated if possible, but external treatment alone is satisfactory if necessary. This treatment is far safer than the radium applicator, which has been severely criticized by L. K. Guggenheim,[8] M. D. Schulz and L. L. Robbins,[9] C. B. Braestrup,[10] I. Lampe,[11] Stacey Guild,[12] Kenneth Day,[13] and others. It has been alleged by proponents of the method that lymphoid nodules in the Eustachian tube itself contribute to impairment of hearing and require this form of treatment. Such nodules, of course, would be surgically inaccessible, but Dorothy Wolff[14] has shown that they are so minute as to be of no possible pathological significance relative to obstruction. Even were this not so, the radium applicator probably could not deliver enough radiation at the necessary distance to affect this tissue. X-ray treatment would be required. A second argument is that minute collections of lymphoid tissue remain in the tunica propria of the vault after even the most complete and meticulous surgical adenoidectomy. Indeed this is true, but what of it? It is a matter for wonder with what fury some feel that these microscopic remnants must be attacked, as though the persistence of a single lymphocyte spelled failure to improve hearing, persistent asthma, postnasal drip, etc. Proper surgery will insure success in the vast majority of cases, and x-ray treatment has been found safe and perfectly satisfactory for the rare case in which surgery alone has not won the day.

SUMMARY

A method of adenoidectomy under control of direct visualization is described, which, properly carried out, will result in a surgically clean nasopharynx.

The surgical inaccessibility of Rosenmüller's fossae is shown to be a fiction.

For the very rare case of conduction hearing impairment in which surgery falls short, the treatment of Lillie, of the Mayo Clinic, is advocated, consisting of the administration of strong iodine solution, U. S. P. by mouth, heat, and pneumatic massage to the membrana tympani. If this is insufficient, x-ray therapy is felt to be far safer than use of the radium applicator.

Cogley Clinic.

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PAINFUL EAR NODULE

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PAINFUL ear nodule is a small keratotic lesion on the edge of the superior and transverse part of the helix. The patient presents himself because of pain severe enough to awaken him at night. Management is difficult because the lesion is usually mistaken for carcinoma, and because recurrence commonly follows removal.

The disorder was originally described in 1916 by Winkler of Switzerland and was designated "chondrodermatitis nodularis chronica helicis." It was named "painful nodular growth of the ear" by Foerster of this country when he independently described the lesion two years later. Subsequent reports are practically limited to dermatological and roentgenological journals, mostly in foreign literature.

The histories related by the patients are typically uniform and immediately indicate the diagnosis. A small nodule at the upper edge of the external ear becomes so painful to touch that a hat cannot be worn. Pain awakens him at night when he turns to the involved side. Pulling the crust off the lesion relieves the pain for a few days. Eighty per cent of the patients are men, and 80% of the involved ears are on the right side. Most of the patients are over 40 years of age.

Examination reveals a single small lesion on the edge of the helix posterosuperiorly. It is oval, crusted, hard, well-defined, and measures about 4 mm. in diameter (Figs. 1 and 2). It is raised 1 or 2 mm. If the keratotic crust has been removed, the lesion is umbilicated, and a small amount of serum covers the pink or yellow floor of the crater. The nodule is usually skin-colored, but may be waxy or pink. The lesion seems to be attached to underlying cartilage, and when manipulation is attempted, the patient draws away because of the exquisite tenderness. The location and appearance of this keratotic nodule vary little except that in women it may arise on the antihelix, particularly if the antihelix projects prominently outward. Occasionally they are multiple. The untreated nodule remains for months or years.

A diagnosis of hyperkeratosis or basal cell carcinoma is usually made; gouty tophi and tuberculosis are diagnostic possibilities. Simple, limited excision of the lesion commonly results in recurrence, thus apparently confirming a diagnosis of carcinoma. Irradiation, electrocoagulation, carbon dioxide snow, and heat have all failed to cure in more than a small percentage of cases.

The solution to the problem of therapy is found after examining a nodule under the microscope. The pathological changes involve not only the epidermis and dermis but the underlying cartilage as well. Superficial layers of the epidermis are thickened, and the rete pegs are deepened. The dermis contains increased blood vessels, a dense accumulation of lymphocytes, and some plasma cells. Underlying cartilage is necrotic. These are inflammatory, rather than neoplastic, changes.

Swedish investigators relate a high incidence of frostbite preceding the occurrence of the nodule.[1] A number of cases in Brazil [2] and California [3] had no such common etiological factor. A few cases in women, whose hair usually protects this portion of the ear, occurred in milliners whose duties included repetitious trying

Fig. 1.—Location and appearance of painful ear nodule (Case 3).

Fig. 2.—Close-up photograph of typical nodule (Case 5).

on of hats. Trauma to avascular tissues in an age group subject to degenerative changes is probably a factor. The definite cause is unknown.

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2. Mangabeira-Albernaz, P., and Monteiro Sales, F. J.: Painful Nodule of Auricular Pavilion (Hemangioacanthokeratoma), Brasil-med. **58**:128-135 (April 22-29) 1944.

3. Culver, G. D.: Painful Nodule of Winkler and Foerster: Report of Cases, California & West. Med. **31**:414-418 (Dec.) 1929.

Therapeutic measures must eradicate the diseased cartilage. Experiences reported in the literature and this series of cases indicate that removal of only the diseased skin is followed by recurrence. In such an instance the skin will sometimes heal without evidence of nodule, but the pain persists. Removal of the nodule and several millimeters of underlying cartilage by cold knife or by electrocoagulation are reported to be the preferred methods. Irradiation is often unsuccessful, and cancericidal doses entail the risk of skin atrophy. Culver cured several patients in his series by removing cartilage alone. He incised through the nodule, trimmed the cartilage with scissors, and coapted the skin edges with the nodule left in place. Relief of pain and disappearance of the nodule resulted. The best results have followed electrodesiccation with a unipolar needle with the patient under local anesthesia. Skin and cartilage are charred and curetted. Quick relief of pain is obtained. The wound heals during a period of several weeks, and a small dimple sometimes remains. The disadvantages of this occasionally slow, though uneventful, recovery seem preferable to the cosmetic defect following a successful surgical excision.

Fig. 3.—Appearance in Case 1 after amputation resulted in recurrence at ends of scar and over original site.

CASE 1.—R. T. M., a 29-year-old lumber-mill worker, was first seen on June 28, 1949, because of a painful nodule of the right ear that he said he wanted cut off. Office records describe it as a 3-mm., hard, keratotic nodule at the edge of the superior helix, seemingly not attached to perichondrium. It was excised after redness subsided three weeks later, and the pathologist reported two small areas of apparent infiltration and recommended treatment as grade 1 squamous cell carcinoma. Since the lesion had been removed superficially, x-ray therapy totaling 3,300 r was given, a cure with a better cosmetic result than surgery would afford being expected. Factors were 100 kv., 15 ma., 25 cm. distance from the target, no filtration, 1,100 r × 3. Three months after the excision biopsy (six weeks after irradiation) a painful nodule was present in the original location. The patient was hospitalized, and the superior portion of the auricle was amputated on Oct. 20. "No evidence of carcinoma" was found by the pathologist on microscopic study of this portion of the ear. The wound healed well, but six weeks later, on Dec. 5, the patient returned because of ear pain, and three nodules were found, one over the original site, and one at each end of the scar (Fig. 3). He has been seen intermittently since that time. The nodules persist. The skin of the upper part of the auricle is atrophic, and the ear is very sensitive to temperature changes. Later review of the tissue slides revealed the picture typical of painful nodule.

CASE 2.—G. F. W., a 57-year-old rancher, first seen on June 9, 1950, complained of a sore on the right ear of one year's duration. The initial diagnosis was keratosis, possibly basal cell carcinoma, of the right ear. Three x-ray treatments, totaling 1,700 r, were given to the ear

lesion without effect. It was excised on Aug. 15, and the microscopic report was "squamous hyperplasia, especially in hair follicles." The pathologist did not believe this represented a carcinoma. The excision was wide enough to clear the lesion by 3 mm., and enough cartilage was removed to facilitate skin closure. The wound healed slowly over a four-week period, the nodule recurred, and the helix remained very tender. The recurrence lay at the anterior end of the scar, not at the original point. About that time the existence of the entity "chondrodermatitis nodularis chronica helicis" was first realized. Electrocoagulation of the recurrent nodule and underlying cartilage was done, and pain was relieved. Some scaling and a small pit remained for several weeks, but there was no pain. When the patient was last seen, on July 23, 1951, a small pit was present at the superior aspect of the helix. There was no nodule, no scaling, and no tenderness.

CASE 3.—J. W. D., an 86-year-old man, was first seen on Jan. 12, 1951. He complained of a painful sore of the right ear. He stated that a similar sore had been present on each ear 15 years before, and that they had disappeared after the application of "acid and iodine." It had now been present on the right ear for a month. His main discomfort came from lying on his right side. Examination revealed a crusted, 2 by 3 mm., raised lesion of the tip of the right helix superiorly. The lesion was umbilicated. Electrocoagulation was done on Jan. 19, and again on Feb. 9. On Feb. 16, he reported the absence of pain and the wound was healed.

CASE 4.—J. P., a 61-year-old innkeeper, was seen on May 8, 1951, because of a painful nodule of the left ear. Examination revealed a 2 by 4 mm., oval, keratotic, umbilicated nodule of the

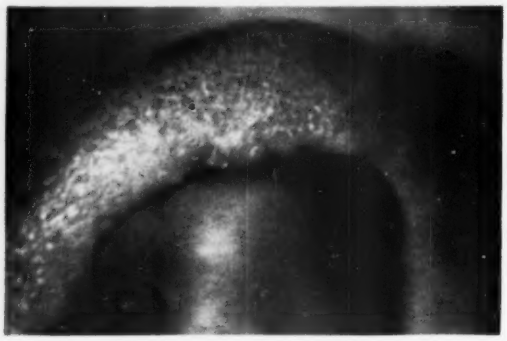

Fig. 4.—Appearance in Case 5 three months after treatment.

free margin of the transverse part of the helix. He did not return for treatment after keeping his appointment with a photographer for a picture of the lesion.

CASE 5.—W. E. D., a 36-year-old heavy-equipment operator for the state highway department, was first seen on May 9, 1951. His right ear had been painful for two months. A typical painful nodule was found at the free margin of the right helix superiorly. After infiltration of procaine hydrochloride, the lesion was electrocoagulated. He was seen again on Aug. 13, when he stated that pain had disappeared on the day of treatment. The wound had remained crusted and slightly infected for eight weeks, then healed completely, leaving a small dimple. The involved area was slightly raised, and some scaling was present, but the hard, discrete, tender nodule was not present (Fig. 4).

SUMMARY

Painful ear nodule is a tender, small, keratotic lesion of the free edge of the superior and transverse portion of the helix.

The appearance, location, and tenderness of the nodule adequately differentiate it from carcinoma.

Immediate treatment by electrocoagulation avoids the deformity and painful recurrences commonly associated with excision biopsy.

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EFFECTS OF ASPIRATION IN THE EXTERNAL EAR CANAL

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FOR UNDISTURBED function, the air content of the cavity system of the middle ear is of utmost importance. A state of balance between the atmospheric pressure and the pressure in the tympanic cavity is indispensable to obtain optimal function of the hearing organ. Valsalva's famous experiment was devised for the restoration of the balance of pressure. However, the original intent of his classic experiment was to utilize it for the expulsion of accumulated pus from the middle ear. Great progress was achieved when it was found that the tympanic pressure could be influenced by a catheter introduced into the pharyngeal ostium of the Eustachian tube.[1] It is almost 80 years ago that Politzer devised his bag for the same purpose. The usefulness of these instruments could not be better proved than through the fact that they are still in use in the identical manner in which they were originally used in spite of the immense technical progress since that time.

Consequently, when the question is brought up: "In what respect has otology not advanced in the last 100 years?" our first answer would be, "The indication and use of the tubal catheter and bag." Nevertheless, even in instances in which correct indication exists and proper technic of these procedures are employed the effect is often far from satisfactory.

The pathological changes causing a disturbed balance of the tympanoatmospheric pressure lead to various severe clinical symptoms.

It is necessary to distinguish between (1) positive balance disorder and (2) negative balance disorder. In positive balance disorder the tympanic air pressure is greater than that of the atmosphere. Its significance is paramount in connection with aviation. Clinical causes of other origin are infrequent. (Actually, the tympanic air pressure is increased with every swallow, but this ventilatory overcompensation is equalized by the tube immediately.) In negative balance disorder, much commoner, the tympanic pressure is lower than atmospheric. The causative factors are generally mechanical in character, and the changes in the middle ear are well known.

The commonest causes of negative balance disorders may be classified as on an organic or functional basis. The organic causes are the following: (1) obstructions of the nasal passages: anatomical anomalies, hyperplasia, accumulation of exudates, and neoplasms; (2) obstructions of the epipharynx: hyperplastic posterior tip of the lower turbinate, adenoid hypertrophy or other local or general lymphatic hyper-

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1. The Eustachian catheter in its present form was introduced into practical usage in 1821 by the French physician Itard.

plasia, tumors, and strictures; (3) obstruction of the Eustachian tube: chronic inflammations, strictures, and adhesions, and (4) obstruction of the middle ear: inflammatory processes, exudates, and adhesions.

The functional causes of negative balance disorder are the following: (1) the well-known effect of sudden change in atmospheric pressure (aero-otitis) (the faultlessly functioning ventilating system will compensate automatically to a great extent; on the other hand, the effect of the atmospheric change usually brings out hidden disturbances of a mechanical character); (2) possible disorder or diminished function of the muscle mechanism of tensor and levator veli palatini which is responsible for proper tubal ventilation.

The symptoms of negative tympanic pressure are subjective and objective in character. Subjective symptoms are (1) diminished hearing, which is always present to a certain extent in all cases of this type (one should note, however, that according to Rasmussen's [2] findings, optimal hearing for speech frequencies is reached at a very slightly negative tympanic pressure, whereas the higher frequencies around 8,000 can be heard better with somewhat increased positive pressure); (2) feeling of fullness in the ear, and (3) tinnitus.

These symptoms may be explained in part by physical laws. When the pressure of the air is changed on one side of a vibrating membrane, its ability to vibrate decreases. Another factor is the influence upon the motion of the ossicles. In addition, the position of the footplate is altered in the oval window, so that even in the resting position it exerts increased pressure upon the perilymph. Finally, in spite of its protected position, the vibration of the membrane of the round window may be influenced. The latter circumstance is probably responsible for a partly or entirely reversible perceptive component in the audiogram in some cases of unbalanced tympanic pressure.

Retraction of the tympanic membrane is one of the objective symptoms of negative pressure. However, this retraction is not the invariable and infallible sign previously supposed. The tympanic membrane may be retracted in the presence of intact hearing and, as shown by van Dishoeck's [3] experiments with the pneumophone, even in cases with normal tympanic pressure. The opposite of this phenomenon is also possible, when, in spite of definite negative pressure in the middle ear, no sign of retraction may be seen. A second objective symptom is found in the various degrees of conductive deafness which are occasionally associated with some sign of decreased perception of hearing in the higher frequencies as a possible consequence of the aforementioned disturbed function of the round window membrane.

As stated above, the catheter and Politzer bag inflation are the oldest procedures employed without modification to this day, and presumably they must be very effective in the control of air pressure in the tympanic cavity. Although this statement may hold true in the vast majority of cases, there are exceptions. It is common experience that after an apparently efficient and well-indicated inflation, the effect is either transient, lasting only a few minutes, or not effective at all. Many attempts have been made to maintain a prolonged effect from inflation.

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Some have suggested a permanent opening in the tympanic membrane. Others have tried applying suction in the external canal when positive pressure from the epipharyngeal end of the Eustachian tube proved ineffective. Cleland, a Scottish military surgeon [4] of the middle of the 18th century, who was the first to utilize a transnasal approach to the pharyngeal opening of the Eustachian tube, advised the following procedure: "In cases of deafness caused by the thunder of a cannon, an exactly fitted tube should be introduced into the patient's external canal. The surgeon puts the other end of the tube into his mouth and creates a suction."

Consequently, we may not state that the idea is a new one, although such an "aspiration" has never become a widely accepted procedure of tubal conditions and is seldom mentioned in the newer textbooks. Politzer [5] describes the procedure of rarefaction of air in the external canal and stresses its great effectiveness in particular cases. The diagnostic as well as the therapeutic value of the procedure is mentioned. The effectiveness of a well-indicated and properly performed aspiration can be observed upon its application.

Aspiration is probably a less physiological procedure than inflation. The latter utilizes the identical pathway of physiological ventilation of the tympanic cavity and at the same time establishes the restoration of this direct connection. Aspiration does not act upon communication. It is an indirect procedure, influencing tension only secondarily and to a smaller degree. Consequently, it is less effective than inflation. On the other hand, aspiration has its effect directly upon the tenseness of the tympanic membrane, on the ossicles, and on the altered adjustment of the footplate. This effe t can be obtained more readily and safely by acting upon the wide and short external canal, rather than on the complicated and longer pathway from the nostril to the tympanic end of the Eustachian tube. An additional great difference is the fact that instead of the frequently uncertain rale of inflation, there is visual observation of the effect.

Aspiration of air through the external canal is performed in the otological practice for several reasons: 1. It is used in combination with inflation in the external canal for (*a*) determining drum mobility, and (*b*) setting up pneumomassage (Delstanche). 2. It is used after paracentesis for the suction and removal of thick and adherent exudate through the relatively narrow opening of the incision (Tobey,[6] Hoople,[7] and others). 3. It is used for the determination of actual tympanic pressure with van Dishoeck's pneumophone. By creating a negative pressure in the external canal, van Dishoeck [8] could determine accurately not only the amount of the actual tympanic pressure but the resistance of the obstructed Eustachian tube. Later, the impaired hearing correlated with the negative tympanic pressure was also measured.[9]

4. Cleland, cited by Stevenson, R. S., and Guthrie, D.: A History of Oto-Laryngology, Baltimore, Williams & Wilkins Company, 1949.

5. Politzer, A.: Lehrbuch der Ohrenheilkunde, ed. 5, Stuttgart, F. Enke, 1908.

6. Tobey, H. G.: Acute Catarrhal Otitis Media with Effusion, Tr. Am. Otol. Soc. **36**:51, 1948.

7. Hoople, G. D.: Otitis Media with Effusion: a Challenge to Otolaryngology, Tr. Amer. Acad. Ophth. **54**:531, 1950.

8. van Dishoeck, H. A. E.: Pneumophon und Wiederstandsmessungen der Ohrtrompete bei Fliegern und Caissonarbeitern, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **146**:248, 1939.

9. van Dishoeck, H. A. E.: Negative Pressure and Loss of Hearing in Tubal Catarrh, Acta Otolaryng. **29**:303, 1941.

I employ negative pressure along with routine otological procedures, for both therapeutic and diagnostic purposes. It can be applied alone or frequently as a very effective addition to tubal inflation.

Siegle's speculum was first used to establish negative pressure in the external canal, but this instrument does not have the advantages that may be obtained by the anastigmatic ear magnifier of Brünings, which is equipped with the attached rubber tube and bag. The procedure is easily applied, harmless, and is done under visual control. In accordance with the caliber of the external canal, the largest or the medium-sized speculum of the Brünings set is used. At times, in order to close the external canal tightly, the application of a rubber ring over the speculum is advisable. The negative pressure is created slowly and gradually with the bag, during which time a slow change in the position of the tympanic membrane becomes visible through the magnifying lens. Simultaneously a slight but definite hyperemia appears along the handle of the malleus, but initially the area immediately surrounding the short process becomes reddened (area of the maleolar artery and branches of the deep auricular arising from the internal maxillary artery). When suction is continued, the hyperemia extends along the entire handle and to that part of the skin of the external canal which is connected to the posterior plica (1 o'clock on the left and 11 o'clock on the right side). At this point, the patient—on rare occasions—feels a slight discomfort in the ear for one or two seconds. The occurrence of this symptom, however, causes far less inconvenience to the patient than occurs with Politzeration.

Experiments of Wever and associates [10] have demonstrated that the resistance of the tympanic membrane is considerable against negative pressure applied through the external canal. In one case —89 mm. Hg, in two others —140 mm. Hg pressure was necessary to cause a rupture. Naturally, there is a great difference whether the desired negative pressure is reached suddenly or gradually. When the hyperemic area is increased in shape or intensity or extends completely over the entire handle of the malleus, the decrease in pressure is stopped. The snugly fitted speculum should be removed gently and gradually. At times it is advisable to increase the pressure slightly in the external canal during removal. The hyperemia disappears or is scarcely visible within one to two minutes; only occasionally may it be seen for a somewhat longer period. In case of an extremely wide or peculiarly curved external canal an adequate aspiration is not feasible. The degree of the suction effect can be estimated by the size and shade of hyperemic area, and the force of aspiration should be varied accordingly if and when the procedure must be repeated. I have used aspiration for diagnostic as well as for therapeutic purposes for years without observing any undesirable sequelae or injury to the tympanic membrane.

The preexisting cardinal symptoms of negative tympanic pressure are definitely influenced by the aspiration to such a marked degree that diagnostic consequences can be drawn from the absence of the effect. The feeling of fullness in the ear ceases immediately—although possibly temporarily—after the first slight aspiration, and the hearing becomes clearer. The tinnitus ceases, or at least decreases in intensity, at once.

10. Wever, E. G.; Lawrence, M., and Smith, K. R.: Effects of Negative Air Pressure in Middle Ear, Ann. Otol. Rhin. & Laryng. **57**:418, 1948.

To evaluate the diagnostic and therapeutic advantages of aspiration, the indications of this very simple procedure may be summarized as follows: Aspiration may be used as a diagnostic procedure: (1) an improvement, small or large, in hearing will set in after aspiration when the loss of hearing—at least partially—is conductive in character, i. e., of middle ear origin; (2) in case of tinnitus the intensity of the noise decreases significantly without exception for a short or long period, or possibly stops entirely after aspiration when it is caused by middle ear involvement.

We possess more exact and simpler methods of determination of a hearing loss of the conductive type, but, among others, aspiration enables us to ascertain the possible association of the middle ear in cases of mixed type of deafness before audiometry or tuning fork tests are done.

The otoscopic picture is not reliable for a satisfactory diagnosis of tubal obstruction. According to Perlman,[11] only the effect of tubal inflation is the diagnostic criterion. One may add to this statement that the objective and subjective improvement following aspiration is a more definite sign of decreased aeration in the tympanum.

The determination of the origin of the tinnitus is of great significance. Tinnitus, which may be influenced partially or totally by tympanic, i. e., meatal, pressure, is based upon pathological changes of the middle ear. Accordingly, we may easily differentiate by aspiration between the two possible origins of tinnitus. Just as the relation of air and bone conduction serves as a key to the evaluation of both groups of hearing loss, similarly aspiration furnishes us a key to the evaluation of the origin of tinnitus. Fowler[12] distinguishes between vibratory tinnitus caused by some sort of actual vibration and nonvibratory tinnitus, generally originating in neuropathological disorders. By an analogy with the classification of the main types of deafness, one may classify tinnitus according to its pathogenesis as "perceptive" and "conductive," depending upon the result of aspiration.

The therapeutic effect of aspiration is likewise significant: 1. The syndrome of tubal obstruction, i. e., negative tympanic pressure, will be affected immediately. When aspiration is performed on only one occasion the improvement is usually transitory (from a few minutes to several days); in the meantime, other routine measures may be applied. 2. When conductive tinnitus and deafness cannot be satisfactorily managed by tubal inflation, aspiration provides an excellent therapeutic effect. In these instances aspiration should be performed alone or in combination with inflation. Results seem to be better when aspiration precedes inflation.

These effects of aspiration may be understood by the simple physical explanation that the function is improved through the creation of negative pressure in the external canal identical with that of the actual pressure in the middle ear. On the other hand, by aspiration the previously decreased tympanic pressure will be further reduced, yet function remains improved during and after the procedure. It may be presumed that this increased negative pressure in the tympanic cavity is able to affect the insufficient tube to a certain degree by its suction effect, and a subse-

11. Perlman, H. B.: Problems in Diagnosis and Evaluation of Treatment of Chronic Tubal Obstruction, Laryngoscope **58**:91, 1948.

12. Fowler, E. P.: Nonvibratory Tinnitus: Factors Underlying Subaudible and Audible Irritations, Arch. Otolaryng. **47**:29 (Jan.) 1948.

quent aeration of the middle ear will set in. This hypothesis is based upon the findings of van Dishoeck that in cases of tubal insufficiency the aeration of the middle ear takes place only through suction effect of tympanic negative pressure. He found, for example, that an actual pressure of —30 mm. Hg in the tympanic cavity will remain constantly at the same level. After a most efficient inflation, the air pressure will decrease gradually at a rapid rate until the previous —30 mm. Hg pressure is reached, and the pressure will not decline further.

Finally it must be mentioned that in cases in which scars or secondary membranes are found on the tympanic membrane, the technic of aspiration must be more careful and gentle, and friable atrophic or secondary membranes will contraindicate the procedure.

SUMMARY

The importance of a well-balanced tympanoatmospheric pressure is stressed.

A positive balance disorder, or a relatively increased tympanic pressure, occurs mostly in aviation. A negative balance disorder, or relatively lower tympanic pressure, is usually a consequence of some mechanical change in the epipharyngeal-tubal pathway.

Aspiration of air in the external canal is described as a safe procedure for the improvement of a negative balance disorder.

Aspiration can be performed alone or as an addition to the effectiveness of routine inflation therapy. A presumed biophysical explanation of the action is given.

From the invariably constant effect of aspiration upon the ill-ventilated middle ear, two diagnostic deductions can be drawn: 1. An improvement in hearing after aspiration calls for—at least partially—a conductive component in the hearing loss. 2. The tinnitus will decrease in intensity, or may cease, after aspiration when caused by middle ear pathological change. Tinnitus remaining unchanged after aspiration is labyrinthine or possibly retrolabyrinthine in origin.

To the extent that the relation of air and bone conduction offers diagnostic aid to pathogenesis of deafness, similarly aspiration may be employed as a pathognostic procedure regarding tinnitus.

By analogy with the two types of deafness, tinnitus may be expressed as conductive, having its origin in the conductive system, and perceptive or sensory, originating through some pathological change in the end-apparatus or pathway of the cochlear nerve.

MASTOIDECTOMY

A New Supra-Auricular Approach to the Middle Ear

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A HORIZONTAL supra-auricular incision above the root of the auricle with a superior approach to the mastoid antrum, middle ear, and labyrinth will be described (Fig. 1). The temporary displacement of the auricle downward and forward requisite for operations upon the middle ear can be obtained by this incision through the scalp at the level of the upper border of the root or attachment of the auricle to the temporal bone.

The auricle with its root is superimposed upon the temporal bone as a shield, or carapace. The root of the auricle is a direct lateral relation of the middle ear, attic, and mastoid antrum and of the upper portion of the mastoid process. Horizontal lines drawn through a section of the ear at the level of the tegmen tympani and tegmen mastoideum (Fig. 2) are in immediate caudal relationship to a horizontal line through the level of the upper border of the root of the auricle—the line of the proposed incision. The actual margin is 1 cm., which permits the incision to be made over temporal bone and obviates any possibility of a postoperative fistula. A further horizontal line through the superior wall of the external auditory meatus marks the lower limit of the attic and antrum. The area thus designated lies directly medial to the upper third of the root of the auricle. The middle third of the root bears a lateral relationship to the middle ear and its contents.

Displacement downward of the root of the auricle to the extent of 2 cm. exposes the temporal bone lateral to the attic, antrum, and middle ear. The cartilage of the auricle is attached to the temporal bone by a more or less connected fibrous mass in which are distinguishable three ligaments, that from the root of the zygomatic arch, that from the upper margin of the porus acusticus externus, and that from the lateral surface of the processus mastoideus. The three auricular muscles, anterior, superior, and posterior, support and give movement to the auricle. Division of the integuments above the pinna and these ligaments permits the auricle to be displaced caudally 1 cm.

The cartilage of the auricle is continued into the cartilage of the external auditory meatus in front and below and forms a half-tube, open above, firmly attached to the lower semicircle of the porus acusticus externus, but loosely connected to the superior wall (Figs. 3 and 4). The cartilaginous trough of the external auditory meatus is completed superiorly by the membranous meatus. That portion of the cartilage of the meatus anteriorly and inferiorly, closely connected to the margins of the bony external auditory meatus, forms the ultimate connection of the root of the

The drawings are by Dr. Clive Stephen, of Melbourne, Australia.

Fig. 1.—The supra-auricular incision.

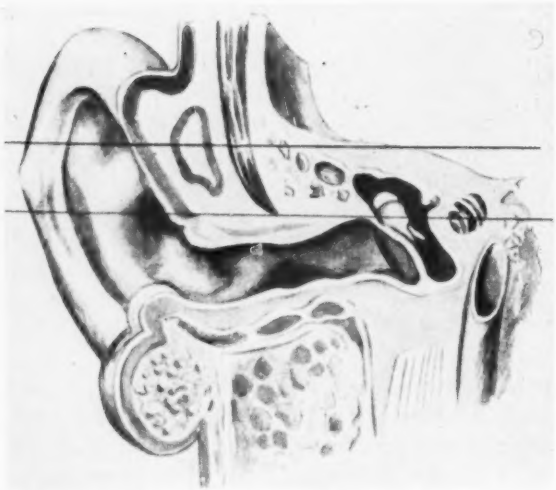

Fig. 2.—Level of auricular attachment and middle ear.

Fig. 3.—Cartilages of auricle and external auditory meatus.

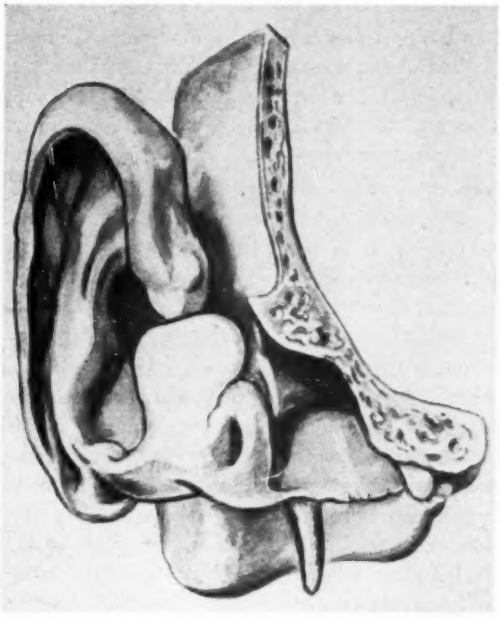

Fig. 4.—Attachment of the auricular-meatal cartilages to the temporal bone.

auricle to the bone of the meatus. The membranous superior wall is loosely connected to the meatus, and the posterior cartilaginous wall is separable easily from the posterior meatal wall.

The root or attachment of the auricle comprehends the external auditory meatus, cartilaginous and membranous, and receives its rigidity from the meatal cartilage and its mobility from the position of attachment of this cartilage.

The integuments and ligaments attaching the auricle to the temporal bone being divided, separation of the superior and posterior walls of the external auditory meatus from the contiguous walls of the bony meatus permits a downward and forward displacement of the auricle and the external auditory meatus. The hinge is the firm attachment of the anterior and inferior portions of the cartilage of the external auditory meatus to the underlying bone of the porus externus.

The posterior incision of Schwartze mobilizes the root of the auricle in this manner. Achievement of adequate access to the attic and middle ear requires an extension of the Schwartze semicircular incision anteriorly above the root of the ear. Division of the superior attachment of the auricle ensues. The posterior segment of the classic incision separating the attachment of the auricle and meatus to the periosteum over the mastoid process may alter the angle which the auricle makes with the skull; the ear may be displaced medially in its projection from the head, with consequent asymmetry with the opposite ear. The division of the superior ligaments and integuments permits of a downward displacement of the auricle, a sagging of the ear and of the external auditory meatus. With an extensive cell development of the mastoid process, the Schwartze incision may not be supported by bone, the incision line is weak, and a postaural fistula may be created.

Abandonment of the posterior segment of the Schwartze incision and extension forward and backward of the horizontal portion would obviate these difficulties. The ear-skull angle is intact; the untouched postaural tissues support the auricle in the original position, and no alteration in the height of the auricle can ensue. No postoperative fistula can appear, as the incision lies upon the temporal bone above the bony excavation.

With the supra-auricular horizontal incision, division of the skin and fascia in the line of the incision and elevation of the periosteum in the lower part of the wound over the mastoid process and attic overhang permit of sufficient retraction forward and downward of the auricle and external auditory meatus to expose the mastoid process completely and permit the performance of every type of mastoid operation.

On the completion of the operative procedure, the wound is sutured. The scar is hidden in the hair of the scalp and by the upward projection of the auricular tip. The fascial attachments of the root of the auricle and the periosteum having been reunited, there can be no dropping of the ear. As the integuments posterior to the pinna have not been divided, no alteration in the angle at which the auricle stands in relation to the skull can appear.

THE OPERATION

General or local anesthesia may be employed. With general anesthesia, infiltration of the operative field with 10 minims (0.61 cc.) of 1:1,000 solution of epinephrine to 2 oz. (59 cc.) of saline solution affords hemostasis and a dry field. With

local anesthesia, a preliminary narcosis is induced with pentobarbital (nembutal®) and "omnopon" (a mixture of soluble hydrochlorides of opium alkaloids). The night before operation 1½ grains (0.09 gm.) of pentobarbital is given. Two hours prior to operation 1½ grains of pentobarbital is administered. One and a half hours prior to operation ⅓ grain (20 mg.) of "omnopon" and 1/150 grain (0.4 mg.) of scopolamine hydrobromide are given. Three grains (0.19 gm.) of pentobarbital is given one hour prior to operation, and one-half hour before operation ⅓ grain of "omnopon" and, if required, a repeat dose of 1½ grains of pentobarbital are administered.

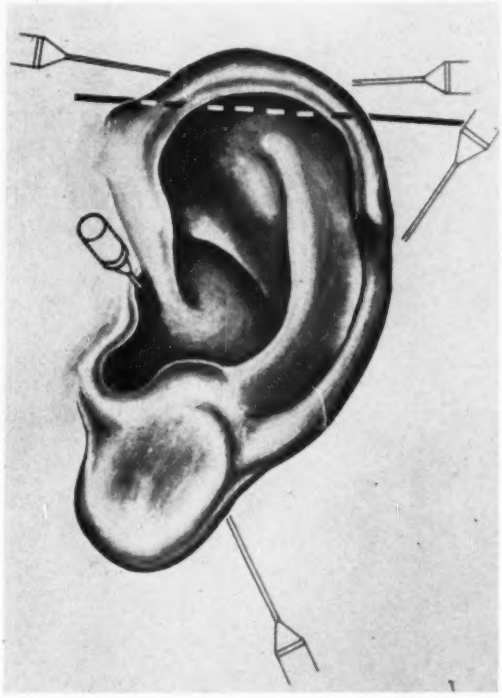

Fig. 5.—Line of the incision and sites of injection of local anesthetic.

The auricle is surrounded with an injection of 2 oz. of 2% procaine solution with 15 minims (0.92 cc.) of epinephrine hydrochloride (1 : 1,000). The incisional area is injected to the periosteum. The mastoid region is infiltrated; 5 cc. of the solution is forced beneath the periosteum over the superior and posterior walls of the membranous external auditory meatus. This ensures hemostasis of the middle ear. An injection is made beneath the ear anterior to the mastoid process. The cavum conchae will be injected when a meatal flap is required (Fig. 5).

The line of incision is outlined, and a dam line of chain catgut sutures inserted to enclose the field of incision. These stitches pass through the whole depth of the integuments and ensure hemostasis. They are removed when the wound is closed.

THE INCISION

The tip of the auricle is drawn downward and an incision made through the scalp immediately above the root of the auricle. It commences at the anterior border of the concha, being limited anteriorly by the superficial temporal artery and

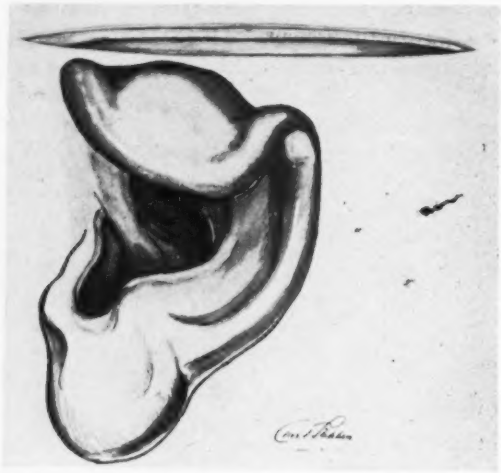

Fig. 6.—The supra-auricular incision.

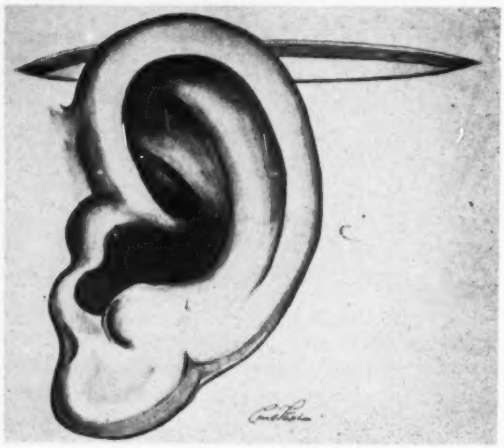

Fig. 7.—Commencement of elevation of periosteum on lower margin of the incision.

vein, and extends horizontally for 2 in. (5 cm.) posteriorly (Fig. 6). The incision extends down to the temporal bone. Bleeding vessels are clamped.

The periosteum is elevated in the lower lip of the wound (Fig. 7), over the mastoid process and the bone superior to the external auditory meatus. The auricle with the lower side of the wound is retracted caudally and forward as the periosteum

is elevated. Self-retaining retractors placed above and below, with a second pair anteriorly and posteriorly, stretch the lower margin of the wound downward and forward. Further elevation of the periosteum exposes the whole of the mastoid process, the superior and posterior surfaces of the external auditory meatus (Fig. 8), and the lip of temporal bone overlying the attic. The whole surgical field for any operative procedures upon the middle ear and its adnexa is divested of its covering of soft parts and lies open and bare within the margins of the wound.

TYPICAL OPERATIONS

Simple Mastoidectomy.—The mastoid antrum is entered with a dental fissure drill, driven inward immediately below and behind the spine of Henle, the outer wall is removed, and the mastoid cells are exenterated with drill, chisel, and ron-

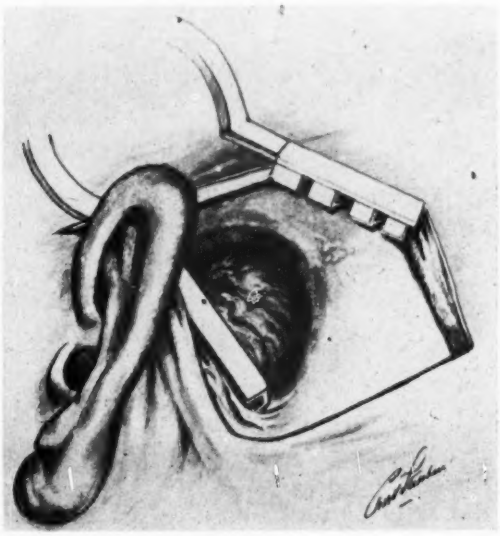

Fig. 8.—Elevation of periosteum and retraction of lower lip of wound, exposing membranous canal and mastoid process.

geurs. Forcible downward retraction of the lower lip of the wound will permit satisfactory access to the most inferior mastoid cells. An extension of the primary incision posteriorly will provide exposure of a lateral sinus in an abnormal posterior location. On completion of the excavation and removal of all infected mucosa, the simple mastoidectomy is complete (Fig. 9). The cavity may be packed, the posterior half of the wound remaining open, and the packing removed or shortened in a few days; or the wound may be closed at once after the insufflation of penicillin-sulfonamide powder.

Atticoantrostomy.—The external auditory membranous canal is raised from the bony meatus on its superior and posterior segments with small elevators down to the annulus tympanicus. The membranous meatal wall will fall away from the bone and be displaced downward and anteriorly by the pull of the lower blade of the retractor. The outer wall of the attic—the overhang—which is the posterior root

of the zygomatic process of the temporal bone, is fully exposed by the displacement of the meatal wall and is removed with the dental fissure drill or chisel, and the attic is opened. The facial ridge is lowered to the level of the tympanic membrane. The annulus tympanicus—the bridge—is thinned from the attic; the tympanic membrane, to which is attached the membranous external auditory canal, is lifted with small

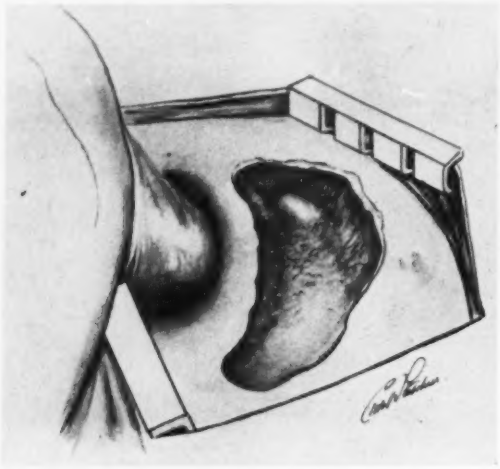

Fig. 9.—Simple mastoidectomy.

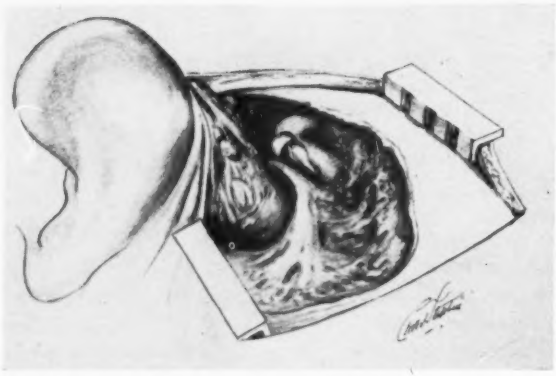

Fig. 10.—Atticoantrostomy.

periosteal elevators, and the bridge broken through with a small curette and removed in its entirety, taking away the anterior and posterior buttresses (Fig. 10). All granulation tissue surrounding the malleus and incus will be removed with a small cranked dental elevator. Necrotic ossicles—incus or malleus head—will be extirpated. The atticoantrostomy, being completed, may be closed without drainage, or the usual tongue-shaped plastic closure may be fashioned from the meatus.

Radical Mastoidectomy.—A further removal of the malleus, incus, and remains of the tympanic membrane, curettage of the mucous membrane of the promontory, removal of the tensor tympani, and possibly curettage of the Eustachian tube will be requisite. No difficulty will arise in viewing the middle ear. The membranous meatus will be ablated and a plastic meatal flap made. The horizontal-supra-auricular incision is closed with sutures.

Labyrinthotomy.—In the fenestration operation the incus is removed; the head of the malleus is amputated. The lateral and superior semicircular canals appear from this approach to be more in the antrum than in the middle ear. The arch of the lateral canal is well shown. The superior canal is entered toward the tegmen antri and is followed down to the vestibule. The lateral canal is opened on the arch posteriorly and along its length to the vestibule. The membranous canals and vestibule are exposed to the maximum extent; the endosteum is removed cleanly.

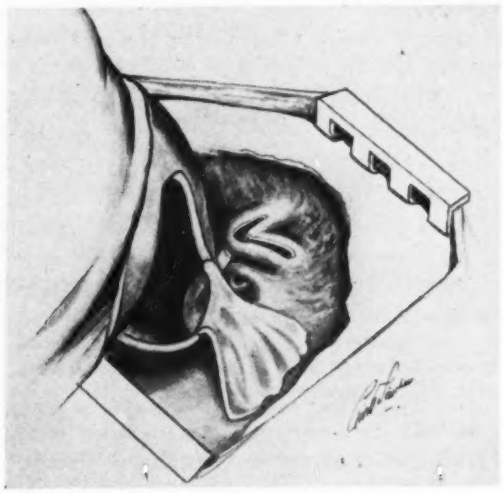

Fig. 11.—The fenestration operation.

The meatal wall is incised and a tongue-shaped flap placed over the labyrinthine fistula. A plastic meatoconchal flap completes the operation (Fig. 11).

Labyrinthectomy.—With the full exposure of the operative field, no difficulty is found in entering the vestibule by breaking down the promontory between the oval and round windows, extracting the stapes and fenestrating the lateral and superior canals.

Closure of the Wound.—The horizontal supra-auricular incision is closed with silkworm mattress sutures, the skin by Michel clips.

SUMMARY

A supra-auricular horizontal incision with a superior approach to the middle ear and its adnexa is described and illustrated by typical operation techniques.

Adequate exposure of the field of operation is provided.

The scar of the incision is invisible in the scalp.

CONGENITAL ATRESIA OF THE AUDITORY CANAL

Two-Stage Operation with Fenestration

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NEW YORK

THE METHOD of treating congenital atresia of the auditory canal has held interest for many years. The studies of Fraser,[1] Altmann,[2] and Richards [3] emphasized the embryological and anatomical anomalies responsible for the condition. Various operators have attempted to make an external canal, which has often helped the hearing a little but has seldom produced improvement up to a serviceable hearing level.

Recently, in 1947, Pattee [4] reported five cases, in four of which there was considerable hearing improvement but still not above the serviceable level in the critical speech frequencies. In a recent discussion of another author's paper he stated that he had since added three cases, in all of which there had been an immediate improvement of hearing but in only one of which the improvement had been maintained.

Early in 1949 Holmes,[5] in an article on the microtic ear, stated:

> . . . Realizing that there is little hope of improving the hearing by methods tried in the past, I should like to see a fenestration operation attempted in one of these cases. There would be no skin flap which could be brought down over the fenestration window, but I am confident that a thin Tiersch graft could be substituted with success.

Rosenberger,[6] in 1949, in his report of an operation in a case of unilateral atresia, obtained hearing improvement but not to the practical level for normal speech reception. In the concluding remarks of his paper he mentioned the possibility of doing a fenestration in such cases.

From the Department of Otolaryngology, Columbia-Presbyterian Medical Center.

Read before the Section of Laryngology, Otology and Rhinology at the One-Hundredth Annual Session of the American Medical Association, Atlantic City, N. J., June 14, 1951.

1. Fraser, J. S., and Davis, E. D.: Mal-Developments of the Auricle, External Meatus and Middle Ear, J. Laryng. & Otol. **44**:544, 1929.

2. Altmann, F.: Zur Anatomie und formalen Genese der Atresia auris congenita, Monatsschr. Ohrenh. **67**:765, 1933.

3. Richards, L.: Congenital Atresia of the External Auditory Meatus, Ann. Otol. Rhin. & Laryng. **42**:692, 1933.

4. Pattee, G. L.: An Operation to Improve Hearing in Cases of Congenital Atresia of the External Auditory Meatus, Arch. Otolaryng. **45**:568 (May) 1947.

5. Holmes, E. M.: The Microtic Ear, Arch. Otolaryng. **49**:243 (March) 1949.

6. Rosenberger, H. C.: Surgery for Congenital Atresia with Report of a Case, Ann. Otol. Rhin. & Laryng. **58**:798, 1949.

Altmann,[7] later in the same year, in a thorough review of over 50 cases of auditory atresia collected from the literature, in which he made a survey of the histological findings, stated that in most of the cases to date sufficient help had not been given as far as hearing improvement was concerned. He suggested a two-stage operative procedure: first, to construct plastically an auditory canal and then, if the hearing is not sufficiently improved, to do a fenestration.

The idea of doing a fenestration on patients in such cases had already been carried out on the Continent by Ombredanne,[8] who in 1947 described two cases of unilateral atresia in children in which he created an external auditory canal and did a fenestration with the use of a Thiersch graft to cover the fenestra, all in one stage. In one of these cases the audiometric result showed speech frequencies above the 30-db. level.

Siirala,[9] in 1949, also reported an operation in a case of unilateral atresia in which he developed a posterior external canal wall with a retroauricular flap, which he carried forward to join the posterior aspect of his endaural incision. The mastoid cavity was then cleaned out; the horizontal canal exposed; a fenestration done, and a Thiersch graft used to cover the fenestra, all in a one-stage procedure. He reported this case as one with an average preoperative loss of 66 db. in the speech frequencies, which were improved by 35 db. postoperatively.

Vogel,[10] in 1949, reported a case in which he used a postauricular approach, doing a modified radical mastoidectomy. He removed the incus and left in place the malformed malleus. He then did a fenestration, covering the fenestra with a Thiersch graft. He stated that there was marked hearing improvement five weeks after the operation but noted a tendency toward stenosis of the cavity.

In January, 1950, there came to me a patient with bilateral atresia who gave me the opportunity to try a two-stage procedure. The result has been so satisfactory that I report it herewith.

REPORT OF A CASE

History.—F. B., a 29-year-old man, was first seen Jan. 18, 1950, at which time a history was obtained to the effect that he had had poor hearing since birth. This was thought to be due to his having been born without external auditory canals. As a result of this condition, his voice was off the normal key and his enunciation of words indistinct. He was said to hear a loud spoken voice and to have a fair ability to read lips. There were no other members of the family with poor hearing or abnormalities of the ear.

Examination.—The patient was a well-developed, husky man of 29. Both auricles were abnormal, the left being more deformed and contorted than the right (Fig. 1). The latter was of nearly normal size, and the appearance was not unduly conspicuous. There was no evidence of an external canal on either side. Roentgenograms of both mastoid areas failed to show any evidence of an external bony canal. Both mastoid bones showed fair pneumatization and small middle-ear cavities. The audiogram (Fig. 2) showed excellent bone conduction across the board for both ears. However, the air conduction, though symmetrical for the two ears, presented a curve showing a loss of 50 to 60 db.

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7. Altmann, F.: Problem of So-Called Congenital Atresia of the Ear, Arch. Otolaryng. **50**:759 (Dec.) 1949.

8. Ombredanne, M.: Chirurgie de la surdite: Fénéstration dans les aplasies de l'oreille avec imperforation du conduit; Resultats, Oto-rhin.-laryng. internat. **31**:229, 1947.

9. Siirala, U.: Plastic Correction of Congenital Atresia of the External Auditory Meatus Combined with Fenestration to Improve Hearing, Acta oto-laryng. **37**:307, 1949.

10. Vogel, K.: Bogengangsfensterung bei angeborener Gehörgangsatresie, Hals-, Nasen- u. Ohrenarzt. **1**:542, 1949.

The patient was advised that he had the choice of being fitted for a hearing aid, a procedure which should be followed with instruction in voice rehabilitation, or of undergoing an operation in an attempt to produce an external auditory canal. If the operation did not bring his hearing

Fig. 1.—*A*, left ear; *B*, right ear, in patient with bilateral atresia.

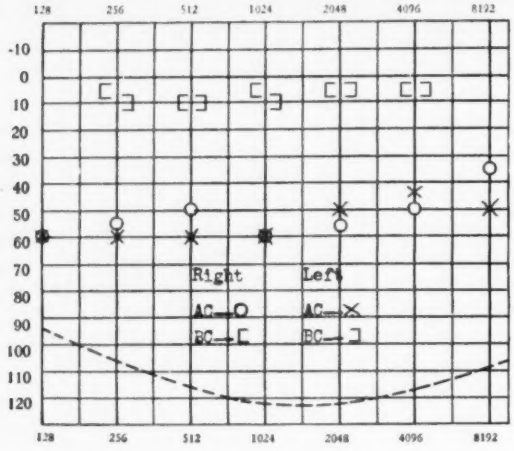

Fig. 2.—Preoperative audiogram (Jan. 18, 1950).

up to a sufficiently high level, it could be followed by a fenestration. The patient refused to consider the wearing of an aid and chose surgery.

First Operation.—On Feb. 3, 1950, endaural radical mastoidectomy was performed. The endaural incision was carried from just behind the tragus upward, well above the level of the

anterior aspect of the pinna. This was carried down to the periosteum. The flaps were retracted, and there was no evidence of bony depression to indicate anything suggestive of an external canal (Fig. 3 *A, B, C*). The mastoid tip was then entered, and the pneumatized cells were found under a thick cortex. The cells were exenterated up to the level of the dural plate, removal being carried deep and forward until the antrum of the middle ear was located. Here an atrophic incus fused with a deformed malleus was located (Fig. 4*D*). These were removed. There was no drum membrane. The mastoid cavity was cleaned out and the exenteration

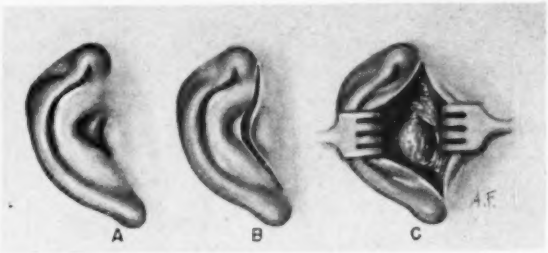

Fig. 3.—*A*, preoperative appearance of right auricle; *B*, endaural incision; *C*, incision down to bone, showing no evidence of an external canal.

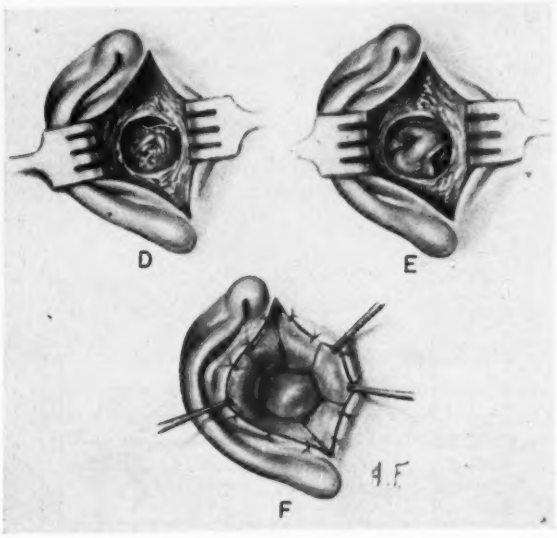

Fig. 4.—*D*, mastoid cells exenterated, exposing malformed incus and malleus; *E*, horizontal canal skeletonized; *F*, placement of skin grafts.

carried forward as far as possible up to the posterior wall of the glenoid fossa. The horizontal canal was identified and well skeletonized (Fig. 4*E*). A large and delicate well-formed stapes was seen, which appeared to be mobile. The entire mastoid cavity was enlarged in such a manner as to make it funnel-shaped, so that there was no overhang at any edge. A skin graft was removed from the abdomen with a dermatome and was cut into six pieces. One piece was cut so as to fit into the depth of the cavity, covering the horizontal canal and extending from the sinodural area forward, to cover the middle ear. This piece was held in place with paraffin mesh gauze. The remaining pieces were placed along the perimeter of the wound and allowed

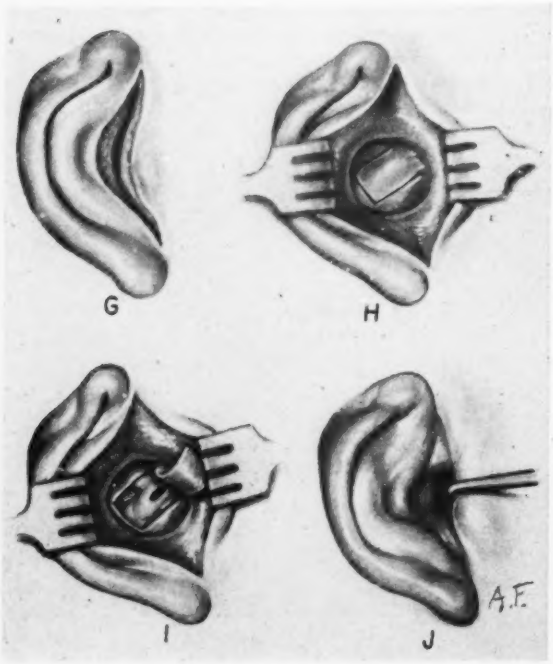

Fig. 5.—*G*, endaural approach; *H*, exposure of cavity and cutting of flap; *I*, fenestration of horizontal canal; *J*, postoperative appearance of external canal.

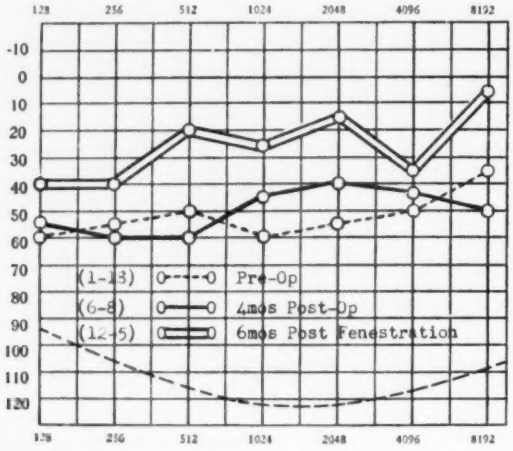

Fig. 6.—Comparative audiograms of right ear before operation and after each operative procedure.

to drop toward the depth of the cavity, and were sutured at the outer edge (Fig. 4*F*). The cavity was then filled with melted paraffin and a dressing applied over the auricle and mastoid area.

The wound was dressed on the sixth postoperative day, at which time the paraffin mold was removed, and from it a duplicate acrylic mold, with a ¼-in. (0.64-cm.) bore hole passing completely through, was made. The latter was then used for about two weeks but not continuously, as the pressure tended to cause sloughing of the skin grafts and the mold had to be kept out one or two days at a time. However, the mold did help in keeping the cavity widened during the early stage of epithelization and prevented the marked contractions that are seen when this precaution is not taken. The cavity, which at first seemed far too large, soon came down to near-normal proportions and yet allowed plenty of room for dressings until all the surfaces were covered with healthy epithelium.

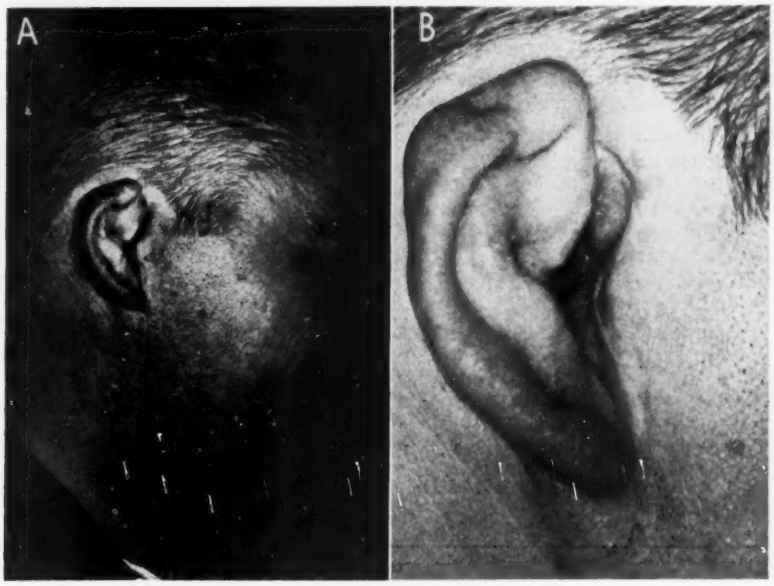

Fig. 7.—*A*, final postoperative appearance of right ear; *B*, close-up of auricle and canal.

During the first few days following the first dressing the patient showed marked hearing improvement, hearing such things as a water tap dripping and sounds which indicated a better-than-serviceable level of hearing. As the weeks passed, however, this early acute hearing soon lapsed to a level about 15 db. better than before operation, leaving him still about 15 db. below a serviceable hearing level in the speech frequencies. I waited until four months had passed and no sign of change or variation had occurred with repeated testing, at which time the patient insisted that the second procedure, namely, the fenestration operation, be tried.

Fenestration.—On June 16, with the patient under thiopental sodium anesthesia, the upper end of the cavity was enlarged via an endaural incision (Fig. 5*G*). This gave large and more than adequate exposure of the cavity. A flap was then cut (Fig. 5*H*), the incision starting at the sinodural end and being carried forward in such a manner as to leave an attached pedicle just anterior to the horizontal canal. The flap was easily elevated with a small periosteal-flap elevator, without any tears or difficulty (Fig. 5*I*). The flap itself was fairly thin and well molded to the area which it had covered and, inasmuch as the horizontal canal had been well skeletonized at the first operation, there remained little to do in this respect except create the fenestra in the usual manner. Practically no bleeding was encountered. The flap was dropped

back into place, and the edges were held in place by paraffin mesh gauze. A very light sea-sponge packing was placed in the cavity.

The postoperative course was the same as in any other fenestration operation except that the reactions of local tissues were less than usual. The flap remained viable. Figure 6 shows the comparative audiograms before and after each procedure. The final gain of 10 db. above the threshold of hearing in the speech range has held up to the present time—one year after fenestration. The final postoperative appearance of the auricle is shown by photographs (Fig. 7).

COMMENT

There are two good reasons for the two-stage operation: First, the plastic creation of an external canal has occasionally produced a good result as to hearing improvement; therefore, it is certainly worth doing the first operation and awaiting the result before going ahead with the second stage, namely, the fenestration. Second, the fenestration flap, as described in the two-stage procedure, is a viable flap, easy to make, well molded to the area to be covered, and involving a minimal amount of tissue reaction, whereas Thiersch grafts, as used in the one-stage procedure, are notoriously poor for the anticipation of a good result in a fenestration. It is not uncommon to have secondary infection in the first operation. This secondary infection may readily spread via the Thiersch graft, causing labyrinthitis and cochlear damage.

SUMMARY

I have described a two-stage procedure for the correction of congenital atresia of the external auditory canal and for improvement of hearing up to the serviceable level. This was accomplished by first plastically creating an external auditory canal and following it later by a second-stage fenestration operation. By so doing, I obtained the use of a viable flap, well molded to the external canal and free of the usual tissue reaction and secondary infection which is present in a one-stage procedure which uses a Thiersch graft.

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ABSTRACT OF DISCUSSION

Dr. George L. Pattee, Denver: Since I have not had any experience with fenestration in these cases, and since this problem is a complex one, if you will excuse me, I am going to give you a little more information that Dr. Woodman did not have time to include.

All these patients do not hear as well as expected after the operation, although theoretically they should have an improvement in hearing. I should like to show you why some of them do not do so well.

It has been my impression, from conversation with otolaryngologists, that many of them do not realize the nature of the embryological deformity. Perhaps it is because of the word "atresia." Dr. Charles Kinney, of Cleveland, suggests that we should not use the word "atresia," because to most of us atresia means a closure of something that was already open. Actually, the definition of "atresia" includes the meaning that no opening was ever present. I agree with Dr. Kinney that many think of atresia of the auditory canal as a canal that was open and then closed, but it never was open and no canal was ever formed.

The first patients whom I operated on obtained immediate hearing improvement, which was encouraging; and then I had some who did not. The question then arises, why do some fail to show improvement? Dr. George Kelemen, of Boston, sent me a slide which shows an abnormal stapes fixed in position by a bony spicule in a case of congenital atresia. It is probably this variation in the congenital deformity of the middle ear that accounts for some of the differences in hearing improvement seen in various cases.

Dr. Lawrence and Dr. Wever have shown that in the transmission of sound waves from air to fluid there is a 30-db. loss of energy. The drum membrane and the ossicles are so arranged

that this 30-db. loss is recovered, and we have normal hearing. Theoretically, then, after a fenestration, and in cases with loss of the membrana tympani and the ossicles, the hearing level with a maximum improvement should be 30 db.

There are probably other factors that allow some patients to get better hearing than others. One is, perhaps, the fortunate accident that allows something to happen in the phase relationship, so that the oval window and the round window move in different phases. A proper phase relationship will raise the hearing acuity after loss of the integrity of the middle-ear structures. Also, there are patients who have better than normal hearing acuity, and some of the best results after fenestration are probably in patients who had a better-than-normal hearing acuity to start with.

I do want to bring up the problem of the fenestration operation in children because of what occurred in one of my cases of bilateral atresia. A year and a half after the original operation, there developed bilateral otitis media, and I was at the Academy meeting at the time. When I got home, I found that someone had opened the middle-ear cavity; so now the patient has constant drainage from one ear and intermittent drainage from the other. If a fenestration is done for this condition in children and otitis media then develops, the possibility of labyrinthitis must not be ignored.

If a fenestration is to be done in these cases, I believe the technique outlined by Dr. Woodman should be followed. I wish to congratulate him on his splendid contribution to otological surgery.

Dr. Edgar M. Holmes, Boston: Dr. Woodman has given us an excellent presentation of the problem of restoring hearing in these most unfortunate persons who happen to be born with bilateral microtia. His case presentation demonstrates that a fenestration in this condition is as practical as it is in otosclerosis.

When I first became interested in this problem, 18 years ago, the slight hope of improving the hearing offered by the literature was a challenge, it being hoped that something that the surgeon might add would be the solution of the problem. In a suitable case, a simple mastoidectomy was performed and the antrum enlarged, exposing the incus. Into this cavity was placed a thin skin graft, which covered the entire mastoid cavity and formed a false membrane over the antrum. The skin graft over this area would bulge on inflation of the Eustachian tube, showing that the middle ear was definitely exposed to sound waves. Audiograms were most discouraging, showing no practical improvement. Further attempts to improve hearing were then discarded until the fenestration operation was perfected, and I discussed with Dr. Philip Meltzer, our Chief of Fenestration Surgery at the Massachusetts Eye and Ear Infirmary, the advisability of employing this procedure on patients with microtia. We concluded that it was definitely worth attempting and that one could anticipate that the hearing would be improved to the extent to which it is in otosclerosis, as the fundamental obstruction to sound after the antrum is reached is the fixed stapes. Between the time of our discussion and our first potential case, Dr. Pattee's article appeared, presenting the logical solution to the problem. The stapes is fixed by the fused and immobile malleus and incus. Remove this impediment, and the motion of the stapes is reestablished. It is as simple as that. Since it is so simple, we reevaluate our processes of thinking and deduction and compliment Dr. Pattee on his contribution to this problem.

To facilitate discussion, a brief review of the embryology underlying the abnormal anatomy that is encountered in the problem is in order.

1. The external auditory meatus and auricle develop from the first branchial groove. Failure to develop gives rise to the deformed auricle and absence of the auditory canal. 2. The malleus and incus are differentiated from the first branchial arch. Failure to differentiate gives the fused ossicles. 3. The stapes is derived from the second arch, independent of the other ossicles, and is found to be normal. 4. The drum membrane is formed by a thinning out of the mesenchymal tissue in the region where the external auditory meatus abuts on the wall of the tympanic cavity. There being no canal, bone is found overlying the entire middle ear, and the drum membrane, of necessity, is absent. 5. The inner ear, or cochlea and labyrinth, is derived from the auditory plaque of ectoderm, which invaginates. Being of separate origin, it is found deformed only when something goes wrong in its development.

Other common abnormalities are as follows: 1. The facial nerve may run in its normal anatomical course or take a course from the geniculate ganglion between the horizontal canal and the stapes; then, instead of going straight through the stylomastoid foramen, it runs in a circle anteriorly, following the location expected of the annulus tympanicus, and makes its exit from the bone apparently in the glenoid fossa. The nerve itself may be smaller than normal. 2. Pneumatization of the mastoid varies from none to complete. 3. Hearing is usually below the 50- to 60-db. level.

What has been found experimentally in animals, and what can be expected of our surgery?

If we close the round window or fix the stapes, an anticipated loss of 50 to 60 db. is obtained. If the drum and ossicles are removed without damaging the stapes and the round window is free of obstruction, a hearing level of plus or minus 30 db. is expected.

With these facts in mind and with the cochlear function normal, we can hope to improve the hearing in cases of microtia to about the 30-db. level, if the middle ear is exposed and the fixed, fused malleus and incus are removed, thus freeing the stapes.

The ideal case for operation is the one in which there are normal bone conduction and a well-pneumatized mastoid. In such a case, it is an easy matter to find the pneumatized mastoid, expose the antrum, and remove the bone anteriorly over the entire middle-ear space, so that when the tympanic plate is thinned to the middle ear, the membrane that lines the space corresponding to the inner surface of the drum membrane is exposed. When this is removed, the deformed and invariably fused malleus and incus may be seen and removed, thus exposing the promontory and the stapes. It is essential to bring the neck of the stapes into prominent view, and to do so it may be necessary to thin down the facial canal and remove Stakes's spine before obtaining an adequate exposure. It is imperative not to damage the membranes of the middle ear, particularly around the stapes and round window, for to do so invites bleeding, scar-tissue formation, and secondary closing of the round window or fixation of the stapes. A thin skin graft may now be placed over the entire area, in the procedure described by Dr. Woodman, being sure that it rests on the stapes. If the hearing fails to improve with this procedure, Dr. Woodman's discussion shows us that all is not lost, and we may expect a hearing improvement by performing a fenestration.

I do not believe that patients with a unilateral atresia should be operated on when the hearing is normal in the other ear. One can expect to improve the hearing up to the 30-db. level only, and that does not materially benefit the patient. If a patient requests operation, understanding the outcome, that is different. One of mine did this, as he wished to enter the armed services, having been refused because of decreased hearing in one ear. After operation he passed the required tests.

I have attempted to operate on microtic ears with absence of pneumatization, and I advise against it. I tried this operation in two cases, using the glenoid fossa as the anterior boundary and the tegmen as the superior one, knowing that somewhere behind the sclerosed bone was the lateral sinus. Bony dissection was carried right in to a point where I thought the tympanic plate should be. When I did not find the middle ear, knowing from other dissections that the facial nerve frequently takes the abnormal course described, my temerity turned to timidity and I stopped short of my goal. The abnormally placed facial nerve has been encountered three times, twice in well-pneumatized bones.

When it seems inadvisable to operate, I have fitted the child to a hearing aid and have found that speech, under guidance, develops well, so that he is able to continue in normal school activities.

Dr. Frederick R. Guilford, Houston, Texas: I have had experience with five cases of so-called congenital atresia of the external auditory canal. My findings have been slightly different from those of Dr. Holmes and Dr. Pattee. I think that we ought to individualize these cases more than the authors have indicated. We must remember that the stage of development of the ear may cease at any point. We must not assume that every patient has deformed ossicles or fused ossicles or fixation of the stapes.

The first patient I operated on had bilateral atresia of both ears and the level in one ear was approximately 40 db. and the level in the other ear approximately 55 db. Both ears were operated on in this patient, and in one ear the bony atresia was not complete. There was a thin

cord of tissue that went down to the level of the drum, and at this point a bony plate was found. This was the ear with better hearing preoperatively. In the other ear there was a complete bony atresia and this ear had a 55-db. level. Beyond the bony atresia there was a chamber that was lined with skin and, again, a plate of bone in place of the tympanic membrane. Both of these ears had very slight deformity, if any, of the ossicles. There was a functioning joint between the malleus and the incus. The malleus was fused to the bony plate which was in the position of the drum membrane. With this in mind, the ossicles were not removed and a pedicle flap was taken from back of the ear. The flap was put down into the cavity over the area of the antrum and the exposed ossicles, and the postoperative result in the ear with the 40-db. level came up to about the 18-db. level and in the other ear it came up to about the 25-db. level. This hearing has been maintained over three years.

In another case we got a gain of about 30-db., giving us a 32-db. level, again leaving the ossicles in place, and from all appearances the ossicular joints were functioning.

The last patient that I operated on has not had a postoperative audiogram but, again, there was not a complete bony atresia. There was a cord of tissue present in the canal and then the depth of the canal appeared to be practically normal. The drum did not appear to have the normal landmarks, however. Nothing more was done than opening up the canal. I think it is important for us to realize that each one of these cases must be individualized and that the appropriate operation must be done. I would certainly agree with Dr. Holmes that I do not believe that patients with unilateral hearing loss should be operated on if the hearing is normal in the other ear, because the gain is not as a rule up to the zero line and we don't offer the patient enough and may jeopardize his health in the future.

DR. DEGRAAF WOODMAN, New York: I thank Drs. Pattee, Holmes, and Guilford for their very interesting discussion.

Dr. Pattee's remark about the procedure not being as simple as it sounds is quite true. Every one of these cases is an individual problem. Our patient was fortunately one who had a good cosmetic and functional result. The results do not always turn out that way.

I was recently called to assist a colleague who had attempted to treat a child in a case of bilateral hearing loss and an immediate facial paralysis followed. On operating the following day, he found the facial nerve to have gone perpendicularly up to the dura without its usual bend under the horizontal canal. This anatomical hazard is the strongest argument for not attempting this work on patients in cases of unilateral loss.

I am pleading for the fenestration only in those cases where needed and, when it is done, only in a second-stage procedure. To attempt in one stage to plastically construct an ear canal, fenestrate the labyrinth, and cover it with a Thiersch graft, and expect to get a good result is expecting too much. Past experience has proved this to several different operators.

I would not hesitate to do a fenestration on a child because of fear of a labyrinthitis secondary to a possible middle ear infection. While my experience is not great, I have done over 100 fenestrations and I have seen 6 middle-ear infections all the way from a few weeks to several months after fenestration, and all but one of those patients had no deterioration in cochlear function.

The criterion as to whether to do a fenestration or not may depend on whether or not we find a mobile stapes. If mobile, then I think we can hope for a good result without doing the operation; but on the other hand I think that the placing of the skin graft, in itself, may help to tend to develop adhesions which cause a fixation of the stapes, and when that condition develops and the hearing decreases, as it has in a great many of these cases a few months later, then I think we should do a fenestration. Therefore, it is a good idea when you do the first procedure to skeletonize the horizontal canal well so that it is easy to raise the flap over the canal and thus make the fenestration a relatively simple matter.

INTRANASAL ENCEPHALOCELES

Survey of Problem, with Recommendations for Reducing Mortality

EXUM WALKER, M.D.
WILLIAM W. MOORE, M.D.
AND
JAMES R. SIMPSON, M.D.
ATLANTA

THE PRINCIPAL purpose of this presentation is to call attention to the diagnostic signs and the proper treatment of intranasal encephalomeningoceles so that they may be recognized and the prevalent excessive mortality greatly reduced.

Although the condition is said to occur very rarely, it seems probable that its incidence is greater than has been realized. Undoubtedly many encephalomeningoceles have been erroneously thought to be nasal polyps or tumors. Furthermore, the high mortality which has followed primary intranasal operations must have discouraged any desire to report these cases. Intranasal encephalomeningoceles comprise about 10% of the total groups of congenital hernias of the cranium. They occupy the nasopharyngeal cavity and communicate with intracranial structures through the lamina cribrosa.

The hernial sac frequently fills the nasopharyngeal space and causes respiratory obstruction. Cerebrospinal rhinorrhea often occurs spontaneously, and it almost invariably follows whenever surgical removal or a biopsy is carried out through the nose as the primary procedure. This leakage predisposes to recurring attacks of meningitis, and the mortality rate has been exceedingly high according to the reported cases in the literature.

The diagnosis is not difficult if the lesion is kept in mind. Any nasal tumor discovered in a newborn baby or a child is apt to be an encephalocele, since polyps and neoplasms are very rare in children. The pedicle always leads to the superior limit of the nasal sulcus. The mass itself often protrudes from one nostril and may be visible in the pharynx. It is soft and compressible and may weep cerebral fluid. Though it may be cystic, aspiration is hazardous because of the risk of meningitis. Rhinorrhea is frequently evident, and the root of the nose may be distended by the intranasal mass.

Roentgenograms of the skull rarely show any defect in the cribriform region, but the nasal septum can be displaced, and the ethmoid cells may appear hazy or absent.

We have found a total of 37 cases which have been published. In 1947 Gisselsson [1] reviewed 33 cases previously reported and added a case of his own. In

From the Neuroclinic, Atlanta.

Presented at the Southern Neurosurgical Society, New Orleans, Feb. 10, 1951.

1. Gisselsson, L.: Intranasal Forms of Encephalomeningocele, Acta Oto-laryng. **35**:519-531, 1947.

these cases, nine patients had associated anomalies and died. Of the remaining 25 patients, 15 died, most of them from meningitis. Of the remaining 10 patients, 5 had rhinorrhea, and in 3 of these meningitis developed. One had a single attack, but the other two had meningitis on repeated occasions.

The intranasal tumor of 13 of the 34 patients was surgically removed. Six of these died of meningitis, and one died after a secondary attempt to control the rhinorrhea by an operation through the frontal sinus. Of the six that survived surgery, there was no record of the ultimate result in two. One patient had meningitis following surgery but was alive one year later. Another had three separate attacks of meningitis within a few weeks after intranasal surgery. The rhinorrhea in this patient was ultimately controlled by an operation through the frontal sinus, but meningitis recurred one year later. One patient was said to have been cured after a secondary operation in which the tumor stalk was ligated after resection of the upper jaw. In a final case the patient was cured when the postoperative rhinorrhea was controlled by a secondary intracranial operation. This was carried out by German,[2] and the operative technique is described in detail in his original publication. He also reported four additional cases of cerebrospinal rhinorrhea of traumatic origin in which operation was done in the same manner.

Ingraham and Matson [3] reported on 546 patients with meningoceles distributed throughout the central nervous system. Of 84 encephaloceles, 5 protruded intranasally. They reported in detail the case of one patient who was successfully treated surgically by exposing the communicating stalk intracranially and repairing the passage through the cribriform plate.

Anderson [4] described a case in which intranasal extirpation of the mass was followed by rhinorrhea and recurring attacks of meningitis. He successfully stopped the rhinorrhea by an intracranial plastic repair of the communication through the cribriform plate.

The case of a premature infant with a massive defect in the ethmoidal region with herniation through a cleft palate was reported by Stuart.[5] An attempt was made to perform an intracranial closure, but the child died.

The fatality reported by Stuart can scarcely be viewed critically, since the patient was a premature infant with a massive herniation and with multiple associated anomalies. We have encountered two other cases of basal encephaloceles of other varieties which were successfully operated upon by Dowman [6] and Dandy,[7] using the intracranial approach.

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2. German, W. J.: Cerebrospinal Rhinorrhea—Surgical Repair, J. Neurosurg. **1**:60-66, 1944.

3. Ingraham, F. D., and Matson, D. D.: Spina Bifida and Cranium Bifidum: IV. An Unusual Nasopharyngeal Encephalocele, New England J. Med. **228**:815-820, 1943.

4. Anderson, F. M.: Intranasal (Sphenopharyngeal) Encephalocele, Arch. Otolaryng. **46**:644-654 (Nov.) 1947.

5. Stuart, E. A.: An Otolaryngologic Aspect of Frontal Meningocele, Arch. Otolaryng. **40**:171-174 (Sept.) 1944.

6. Funkhouser, W. L.: Encephalo-Meningocele Through the Foramen Cecum, South. M. J. **15**:385-387, 1922.

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In our review of the literature we have found a total of three cases in which an intracranial repair of an intranasal encephalocele has been successfully carried out. Our own case adds a fourth to this group.

REPORT OF A CASE

History.—A baby girl, C. H. A., born Feb. 17, 1950, was observed at birth to have a growth protruding from the right nostril. A portion of this was removed with a snare about 12 hours later. The microscopic sections of the material indicated that it was an encephalocele.

The child's general health had been good with the exception that she had had considerable difficulty in breathing because of the nasal obstruction. Clear fluid had been draining from the nose.

One could see a rounded, reddish, soft, tumorous mass distending the nose and protruding from the right nostril. Several drops of clear fluid resembling cerebrospinal fluid were seen exuding from the right nostril about the tumor. The intranasal mass had caused an obvious swelling of the root of the nose on the right side. The left nasal cavity was quite narrow, and the child was not observed to breathe through her nose. On looking into the pharynx, one could see the tumorous mass extending down from the superior pharyngeal region.

Fig. 1.—Cerebrospinal fluid is shown oozing from the surface of the encephalocele. Note also the distension of the nose by the tumor within.

The child's breathing was periodically labored. She would struggle with rapid semi-obstructed respirations until exhausted and then lie motionless in an alternate phase of apnea. Although the respiratory difficulty was not immediately threatening to the child's life, it caused reasonable concern. She seemed otherwise normal, and no other anomalies were disclosed by neurological and physical examinations.

Roentgenograms of the skull showed some obvious deformity in the nasal region. The right nasal cavity was obviously distended by a mass. The septum, which was displaced markedly to the left, almost obliterated the left nasal space, and the outer wall was displaced laterally. The lateral view depicted a shadow filling the superior pharynx and extending downward to the tongue. The cause of the respiratory difficulty was very evident. One could not see any definite defect in the region of the cribriform plate.

Treatment and Progress.—A tracheotomy was immediately performed, on March 13, 1950, and the respiratory distress was relieved.

Penicillin therapy was instituted to control any meningeal infection. The infant took her feedings better, and her general condition improved progressively. After gaining 4 ounces (115 gm.), she weighed 5 pounds, 11 ounces (2,680 gm.).

A craniotomy with repair of a cerebrospinal fluid fistula was done March 20, 1950. Local anesthesia was used along with thiopental sodium intravenously, and this combination worked

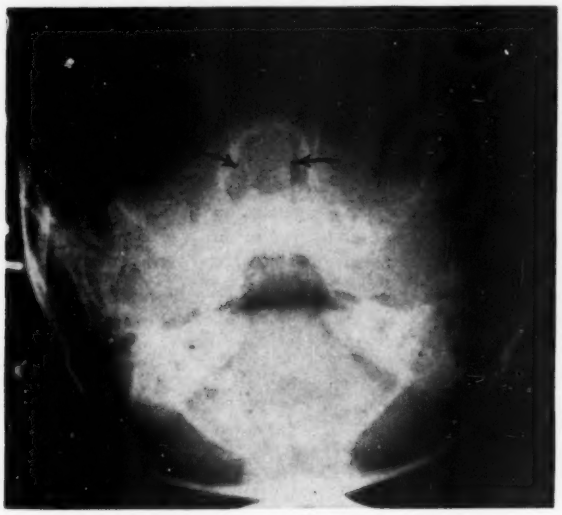

Fig. 2.—The intranasal mass is shown distending the right nasal cavity, whereas the left side is obliterated.

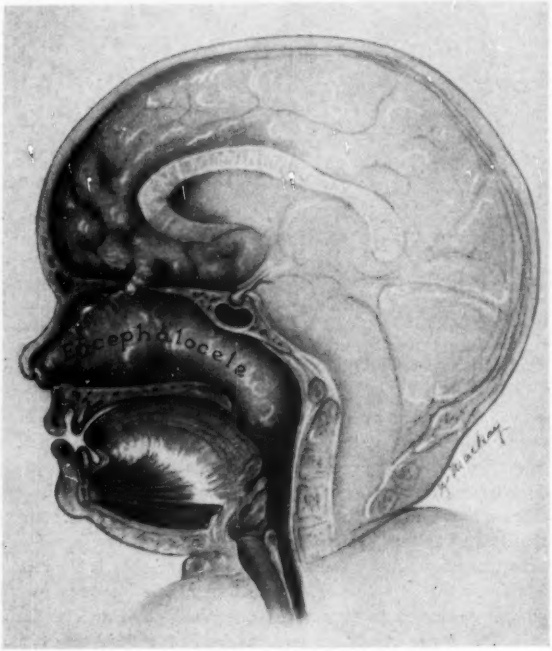

Fig. 3.—Reconstructive drawing to show the relationships of the encephalocele.

very well. The child had only a small amount of thiopental sodium. At the termination of the operative procedure she was moving all of her extremities, and her general condition was excellent. During the procedure 75 cc. of blood was given.

A small right frontal bone flap was reflected, and the prechiasmal region was approached. As the brain was retracted and the orbital plate exposed, one could see a connection between the brain and an oval area occupying the cribriform region. This oval area extended laterally from the base of the cristi galli and measured approximately 7 by 15 mm. The attachment of brain tissue to this region, which was rather vascular but thin, seemed to consist mostly of arachnoidal tissue. After this tissue was coagulated and divided, the oval defect was exposed and the dura could be seen to pass over the rim of this aperture on all sides. A flap of dura measuring about 2 cm. in diameter was obtained from over the orbital plate and reflected

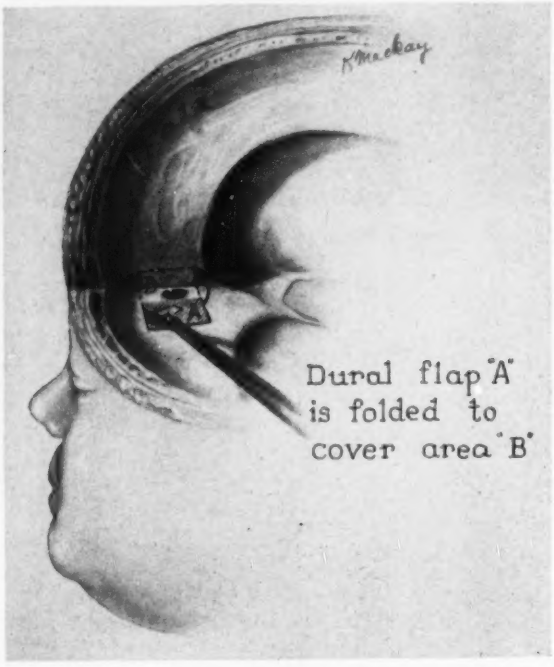

Fig. 4.—Manner of closing over the cranial defect. This is the same technique which we have used satisfactorily in certain cases of rhinorrhea due to other causes.

medially over the oval defect. The opposing surfaces had been scarified to encourage adhesion. This dural flap overlay the defect, extended beyond the margin about 8 mm. in all directions, and folded over the cristi galli and part of the falx. The dura was pressed into place snugly, and a strip of absorbable gelatin sponge (gelfoam®) was placed over the entire region. The frontal lobe was then allowed to fall back into place. I had considered suturing the dural flap but did not feel that this would add materially to the security of the flap. The suture line could not be made waterproof, and the flap was held in place by the absorbable gelatin sponge and the apposition of the brain so that it would not be dislodged. The olfactory bulb was not seen on the right side and was presumed to have been congenitally absent. The dura was closed and the wound repaired. The entire procedure was carried out without any undue technical difficulty. The exposure was adequate, and there was scarcely any loss of blood.

The child was placed in bed with the head elevated to minimize any leakage of spinal fluid.

There was never any further leakage of cerebrospinal fluid. Although the intranasal mass promptly shrank, it still caused enough respiratory obstruction to prevent the removal of the tracheotomy tube.

On June 8, 1950, the nasopharyngeal mass was removed surgically without undue difficulty, and an adequate nasal airway was thus established. Then the tracheotomy tube was gradually occluded and finally removed.

Follow-Up.—The child has developed normally and remained well (Feb., 1951). She has had no leakage of cerebrospinal fluid or any respiratory difficulty and appears to be cured.

COMMENT

The review of the literature indicates that the occurrence of intranasal encephaloceles has been associated with an extraordinarily high mortality. Analysis reveals that this has been due to failure to recognize the nature of this condition and the performance of ill-advised intranasal aspiration, biopsy, or surgery. Meningitis, which has been the usual cause of death, could have been prevented in many instances if adequate chemotherapy had been available. Nasal obstruction has also been a contributory factor.

The mortality rate can be reduced to a negligible minimum if correct diagnoses are made and the cerebrospinal fluid communications are safely repaired. It is apparent from past experiences that any intranasal procedure is dangerous and should be avoided until the intracranial connection has been sealed off. This should be done through an intracranial approach which affords adequate exposure and is performed through a sterile field. This is a relatively simple and safe neurosurgical procedure. Any persisting nasal obstruction may be corrected by an intranasal removal of the hernial mass after the cerebrospinal fluid spaces have been well sealed off from above.

In this case the preliminary tracheotomy was of great value, and it seems doubtful that the child would have survived without it. We therefore recommend its use in the future whenever nasal obstruction is significant.

CONCLUSIONS

Intranasal encephalomeningoceles probably occur much more frequently than is indicated by the number of reported cases.

The principal causes of the extraordinarily high mortality rate in the past have been the failure to appreciate the nature of the lesion and the ill-advised intranasal procedures which have resulted in meningitis.

The mortality should be reduced to an insignificant minimum if the following instructions are observed.

1. Diagnose correctly.
2. Avoid any intranasal procedure which may initiate leakage of cerebrospinal fluid.
3. Institute adequate chemotherapy.
4. If any significant nasal obstruction exists, do a tracheotomy.
5. Perform an intracranial repair of the cerebrospinal fluid communication as a primary operation.
6. If there is any persisting nasal obstruction, relieve it by an intranasal removal of the hernial mass.

133 Doctors' Building (3).

NOTES ON THE INNERVATION OF THE COCHLEA

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THE SCHEMATIC representation of the general distribution of the eighth nerve to the organs of the inner ear, which is reproduced in Figure 1, was worked out by Weston in 1937 and closely corresponds to the representation formerly given by de Burlet except for the cochleosaccular ramus, first described by Hardy in 1934. It must be considered the synthesis of the achievements of a formidable work of research carried out for more than a century by all investigators of the anatomy of the ear.

However, some of the problems regarding the origin and peripheral distribution of the fibers of the eighth nerve have not been definitely solved. One of these problems concerns the inferior saccular ramus, whose origin, according to different authors, is identified in an independent ganglion,[1] in a ganglion annexed to the trunk of the cochlear nerve,[2] in an expansion of the vestibular ganglion,[3] or in the vestibular ganglion itself.[4] The direct origin in the vestibular ganglion is generally accepted today.

The second problem concerns the vestibulocochlear anastomosis. A bundle of fibers was first described by Boettcher,[1] having its origin in the ganglion bearing his name, and distributing partly to the initial portion of the cochlear canal and partly to the macula sacculi. A detailed description of this bundle was given later by Oort,[4c] who was of the opinion that it derives from the inferior vestibular ganglion. This opinion was confirmed by successive investigators, among whom was Kolmer.[4a,b] Weston,[4d] on the contrary, believed that it consists mainly of aberrant cochlear fibers which reach the initial portion of the cochlear ganglion, passing

From the Institute of Anatomy (Director, Prof. A. C. Bruni) and the Ear, Nose, and Throat Clinic (Director, Prof. L. Pietrantoni) of the University of Milan.

1. Boettcher, A.: Über Entwickelung und Bau des Gehörlabyrinths, Verhandl. K. Leop. Carol. deutsch Akad. Naturforscher, 1869.

2. Testut, L., and Latarjet, A.: Traité d'anatomie humaine, Ed. 8, Paris, Gaston Doin & Cie, 1931, Vol. 2.

3. Cannieu: Recherches sur le nerf auditif, ses rameaux et ses ganglions, Rev. biol. Nord France **6**:87, 1893-1894. Bonnier, P.: L'oreille, Paris, Masson & Cie, 1896, Vol. 1: Anatomie.

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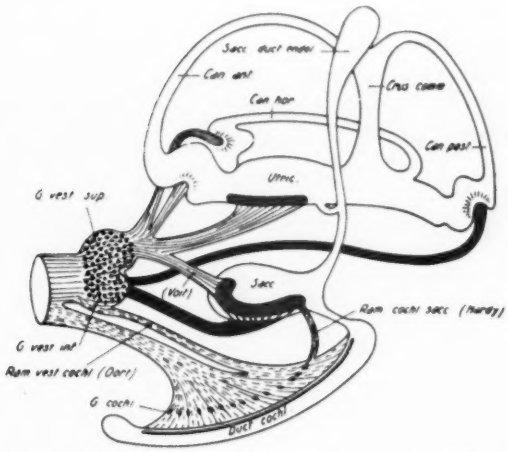

Fig. 1.—Schematic representation of inner ear in mammals (after Weston).

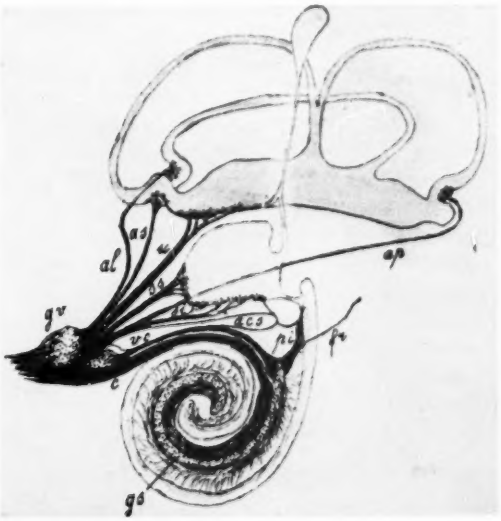

Fig. 2.—Schematic representation of inner ear of rat, according to my observations. *Al*, ramum ampullaris lateralis; *as*, ramus ampullaris superior; *ap*, ramus ampullaris posterior; *u*, ramus utricularis; *ss*, ramus saccularis superior; *si*, ramus saccularis inferior; *acs*, anastomosis cochleosaccularis; *pi*, ramus partis initialis; *vc*, ramus vestibulocochlearis (Oort); *fr*, ramus fenestrae rotundae; *gs*, ganglion spirale; *gv*, ganglion vestibulare.

through the inferior vestibular ganglion or adjacent to it. In man, Mesolella [5] was not able to identify Boettcher's ganglion and the vestibulocochlear bundle.

The third problem concerns the cochleosaccular ramus described by Hardy,[6] which was considered by Hardy [6] and Lorente de Nó [7] to consist merely, like the vestibulocochlear ramus of Oort, of aberrant cochlear fibers. The fourth very much debated problem concerns the arrangement of cells and fibers in the lamina spiralis ossea; it will be dealt with more extensively later.

THE INVESTIGATION

The material used in the present research has been kindly put at my disposal by Prof. A. C. Bruni from his private collection; this includes a vast number of serial sections of the heads of rat fetuses at different stages of development, stained according to the method of Cajal. It has been possible to observe with satisfactory precision the vestibular and spiral ganglia and the course and distribution of their peripheral fibers. The vestibular fibers have been followed up to the cristae ampullares and the sacculus, and the cochlear fibers, up to their entrance into the organ of Corti. The cochlear fibers have been followed also centrally to the ganglion, with the aim of studying their arrangement along the trunk of the nerve in the mod olus. No further study of the nerve fibers in the cochlear canal has been made; however, their arrangement is sufficiently well known after the works of Kolmer [4a, b] and Lorente de Nó,[8] Palumbi,[9] and del Bo and Livan.[10]

The following observations seem to be of importance:

1. An expansion of the vestibular ganglion lies strictly adherent to the cochlear nerve in the base of the modiolus: The conspicuous vestibulocochlear ramus of Boettcher and Oort seems to consist partly of peripheral fibers coming from this expansion (Fig. 3*A* and 3*B*) and partly of central fibers coming from the initial portion of the spiral ganglion; it receives two anastomoses from the inferior saccular ramus, one in the vicinity of the vestibular ganglion and the other somewhat more distally (Fig. 4*A*). The latter appears to be formed by recurrent vestibular fibers disposed in such a manner to recall the arrangement of the recurrent cervical fibers in the ansa hypoglossi. Two twigs take their origin from this anastomotic loop, one reaching the initial portion of the cochlea, where it terminates independently of the fibers coming from the spiral ganglion, the other distributing to the macula sacculi. In my preparations I failed to recognize the cochleosaccular ramus of Hardy; on the contrary, I was able to identify a ramus so far undescribed,

5. Mesolella, V.: Sul cosiddetto ramo vestibolare del cocleare e sul suo ganglio (ganglio di Boettcher), Att. clin. otorin. Napoli **15**:41, 1939.

6. Hardy, M.: Observations on the Innervation of the Macula Sacculi in Man, Anat. Rec. **59**:403, 1934.

7. (*a*) Lorente de Nó, R.: Anatomy of the Eighth Nerve, Laryngoscope **43**:1, 1933; (*b*) Untersuchungen über die Anatomie und Physiologie des Ohrlabyrinthes und des N. octavus, Monatsschr. Ohrenh. **61**:857, 1927.

8. Lorente de Nó, R.: Symposium: Neural Mechanism of Hearing; Anatomy and Physiology; Sensory Endings in the Cochlea, Laryngoscope **47**:373, 1937.

9. Palumbi, G.: Nuovi apparati espansionali nervosi recettori nel labirinto membranoso dell'orecchio umano, Boll. Soc. med.-chir. Pavia **5**:1, 1950.

10. del Bo, M., and Livan, M.: Nuovi dati sulla distribuzione delle fibre cocleari ecc., Boll. Soc. med.-chir. Pavia **5**:1103, 1950; De l'existence d'un plexe nerveus marginal et paramarginal au niveau de la lame spirale, Acta anat. **13**:16, 1951.

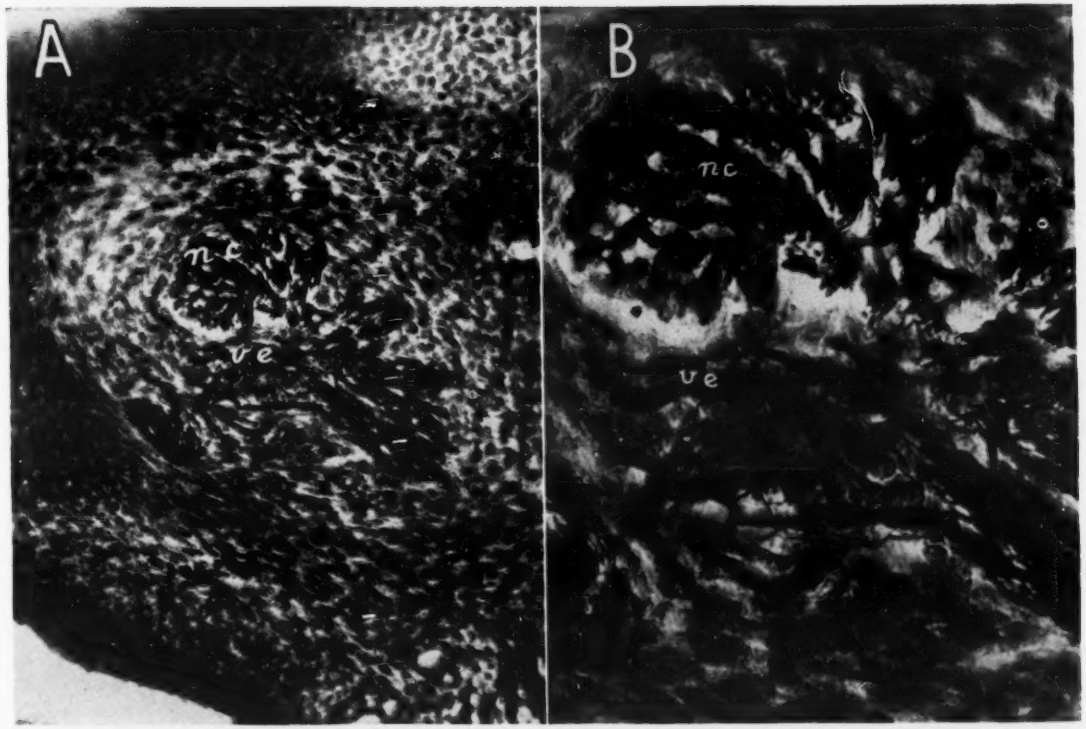

Fig. 3.—*A*, horizontal section through base of modiolus: *nc*, nervus cochlearis; *ve*, modiolar expansion of vestibular ganglion with its peripheral neurons joining the cochlear fibers (× 200).

B, the same at higher magnification (× 520).

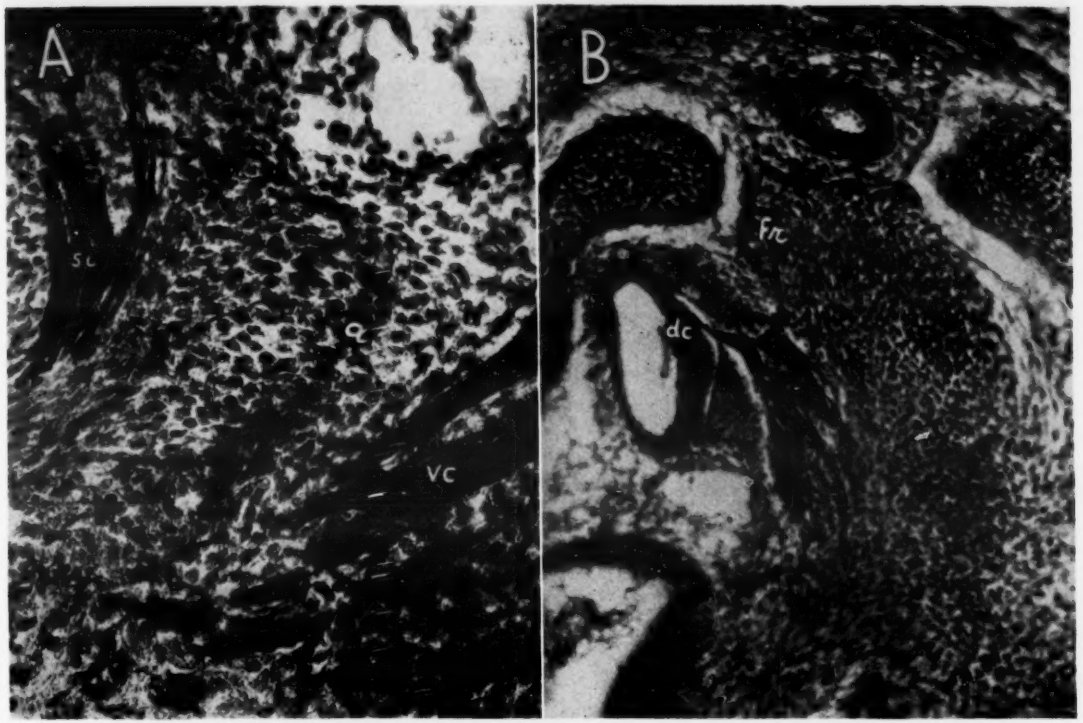

Fig. 4.—*A*, horizontal section through base of modiolus. The anastomotic loop, *a*, between inferior saccular ramus, *si*, and vestibulocochlear ramus, *vc*, is well shown (× 280).

B, horizontal section at level of initial portion of cochlea: *fr*, ramus of round window; *dc*, ductus cochlearis (× 145).

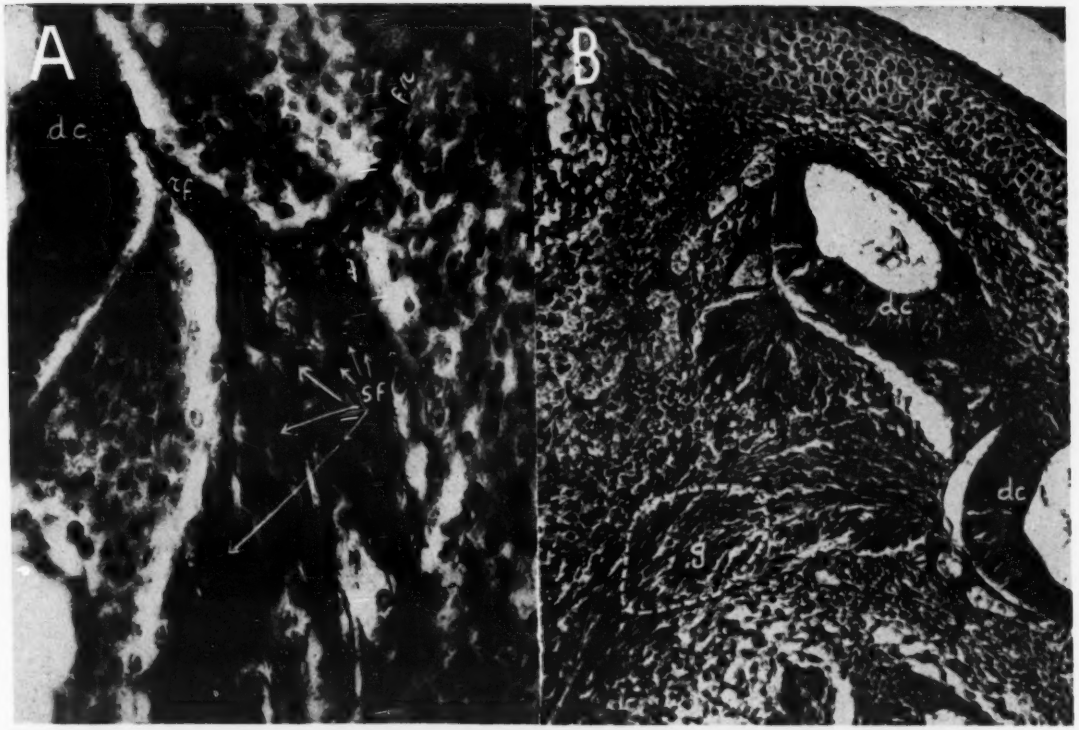

Fig. 5.—*A*, the same as Fig. *4A* at higher magnification: *fr*, radial fibers running to cochlear duct, *dc; sf*, spiral fibers shown as black dots, owing to orientation of section (× 440).

B, sagittal section of cochlea at level of middle coil. Large group of ganglion cells, *g*, contained in modiolus (× 440).

and sometimes double, which takes its origin from the ramus of the initial portion of the cochlea and runs a brief course, soon being lost in the connective tissue of the vestibular wall in the vicinity of the round window (Figs. 4*B*, 5*A*). It is uncertain whether its fibers come from the vestibular or from the cochlear ganglion, but it may represent the continuation of some of the vestibular fibers contained in the vestibulocochlear ramus.

The drawing reproduced in Figure 2 illustrates the general distribution of the eighth nerve according to the results of my observations.

2. The second ganglion described by Boettcher, located in the modiolus among the fibers of the cochlear nerve, was also investigated in our series of sagittal sections of the rat fetus of 19 mm. I was able to confirm its existence (Fig. 5*B*) and to observe that it extends farther than reported by Boettcher, from the vicinity of the expansion of the vestibular ganglion, in the base of the modiolus, up to the level of the initial portion of the apical coil. However, I was not able to confirm Boettcher's view that it is an independent gangliar formation. In my observations this ganglion becomes fused with the spiral ganglion at the beginning of the apical coil; therefore it must be considered as a modiolar expansion of the spiral ganglion.

The ultimate separation of the cochlear from the vestibular ganglion in the course of development takes place presumably between the lower part of this modiolar expansion of the spiral ganglion and the upper part of the modiolar extension of the vestibular ganglion; however, the two formations appear to be already sharply divided in the late stages of fetal life. This modiolar extension of the spiral ganglion tends to disappear in postnatal life, being absorbed definitely into the apical portion of the spiral ganglion itself; however, numerous isolated ganglion cells along the upper part of the modiolus may still be observed in adult guinea pigs.

3. The region of the spiral ganglion has been the object of my special attention, and I have been able, in sections placed parallel to the base of the cochlea, to recognize most clearly two systems of fibers, one running in a radial direction and the other in a spiral direction. The latter was first described by Boettcher and its presence confirmed by other investigators. I shall consider the two systems separately.

SYSTEM OF RADIAL FIBERS

Most investigators agree that the central and peripheral fibers of the cochlear nerve follow an orderly course from the modiolus into the lamina spiralis ossea. It appeared clear from my preparations not only that the fibers are regularly and orderly arranged in the lamina spiralis ossea, but also that the ganglion cells are most regularly and uniformly arranged in radial rows in any stage of development, with their major axis always lying along the same radial direction (Fig. 6*A*). The bundles of central fibers reach the ganglion at regular intervals, and each bundle spreads out in a brush-like formation, making contact with a group of about 10 ganglion cells (Fig. 6*A*). In analogous fashion, the peripheral neurons collect, converging into bundles which, in properly orientated sections, appear to be formed of about 10 fibers each (Fig. 6*B*) and to correspond in number to the foramina nervosa (foramina of the habenula perforata). The observation of a plexus-like arrangement of the cochlear neurons, reported by some investigators,[4a,b] is clearly to be ascribed to the superimposition of different planes at low magnification, including the plane of the spiral fibers, which I shall describe later (Figs. 6*A*, 6*B*,

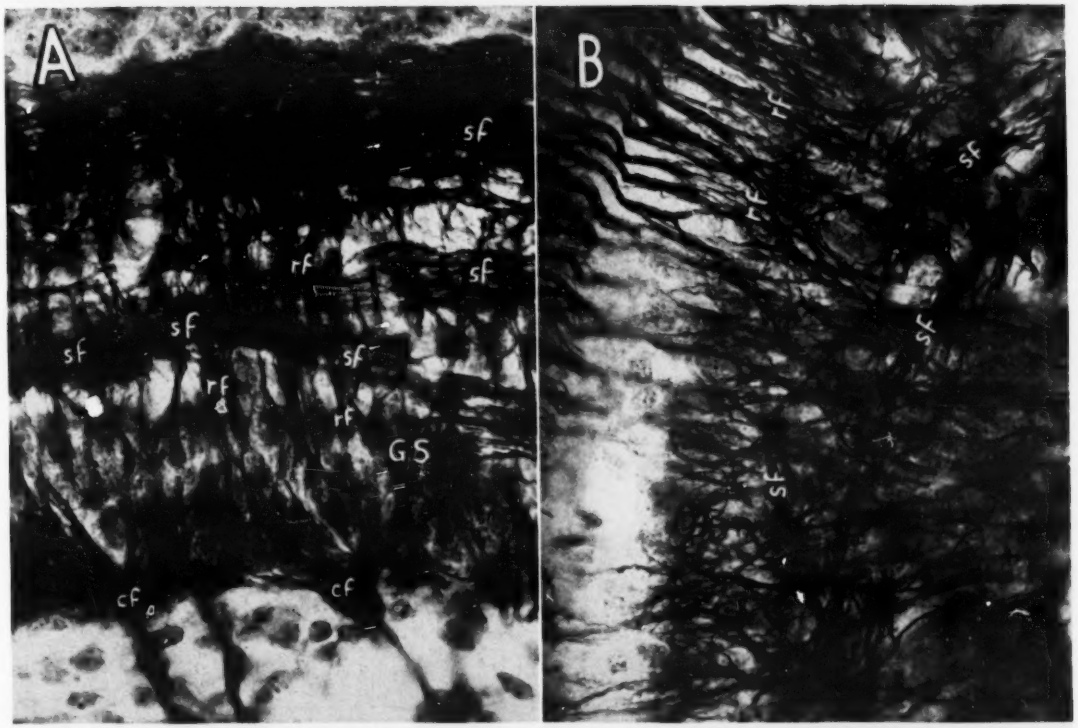

Fig. 6.—*A*, horizontal section through lamina spiralis. The bundles of central fibers enter spiral ganglion, *GS*, at regular intervals, spreading out in brush-like formation. The strictly radial arrangement of cells and fibers is evident, *rf*. Spiral fibers, *sf*, may also be seen faintly, crossing radial fibers, *rf*, on a different plane (× 700).

B, horizontal section through lamina spiralis, middle coil. Grouping of peripheral neurons, to form bundles which enter the foramina nervosa (habenula perforata) is clearly evident. Many spiral fibers, *sf*, cross the radial fibers, *rf*, mainly on a different plane (× 700).

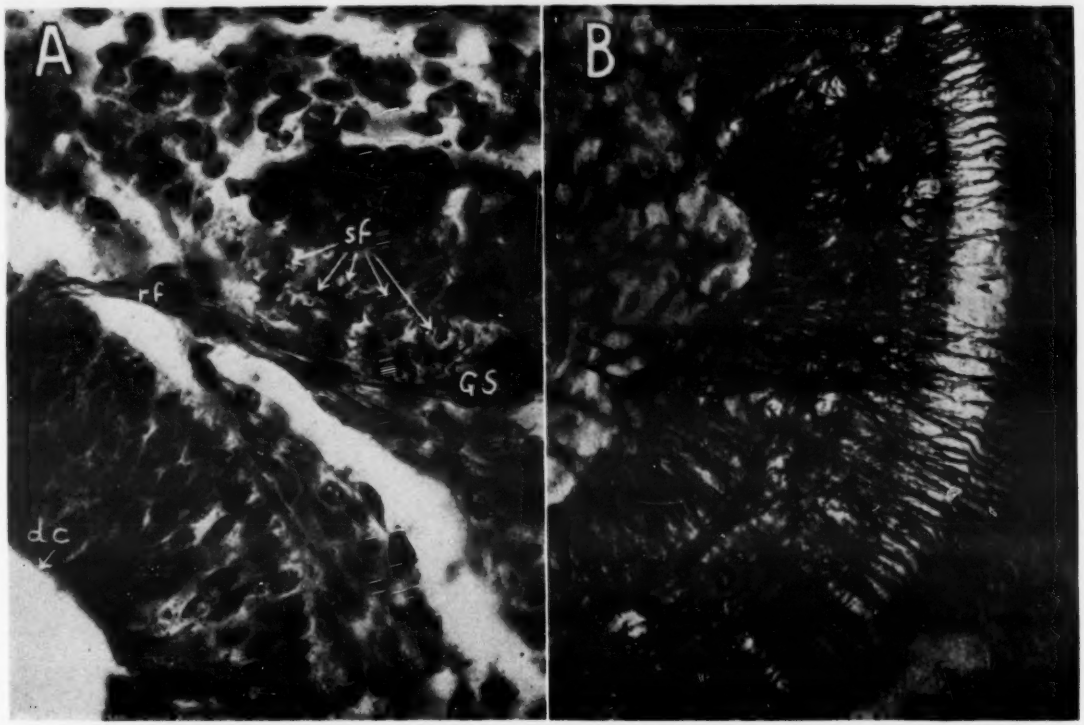

Fig. 7.—*A*, sagittal section of cochlea, parallel to axis of modiolus, middle coil: *dc*, ductus cochlearis; *GS*, ganglion spirale; *sf*, spiral fibers cut transversally and appearing as black dots (Fig. 5*A*); *rf*, radial fibers entering cochlear duct through the foramina nervosa (× 750).

B, horizontal section through lamina spiralis, middle coil. Superimposition of different planes due to weak magnification gives appearance of a plexus-like arrangement of ganglion fibers (× 320).

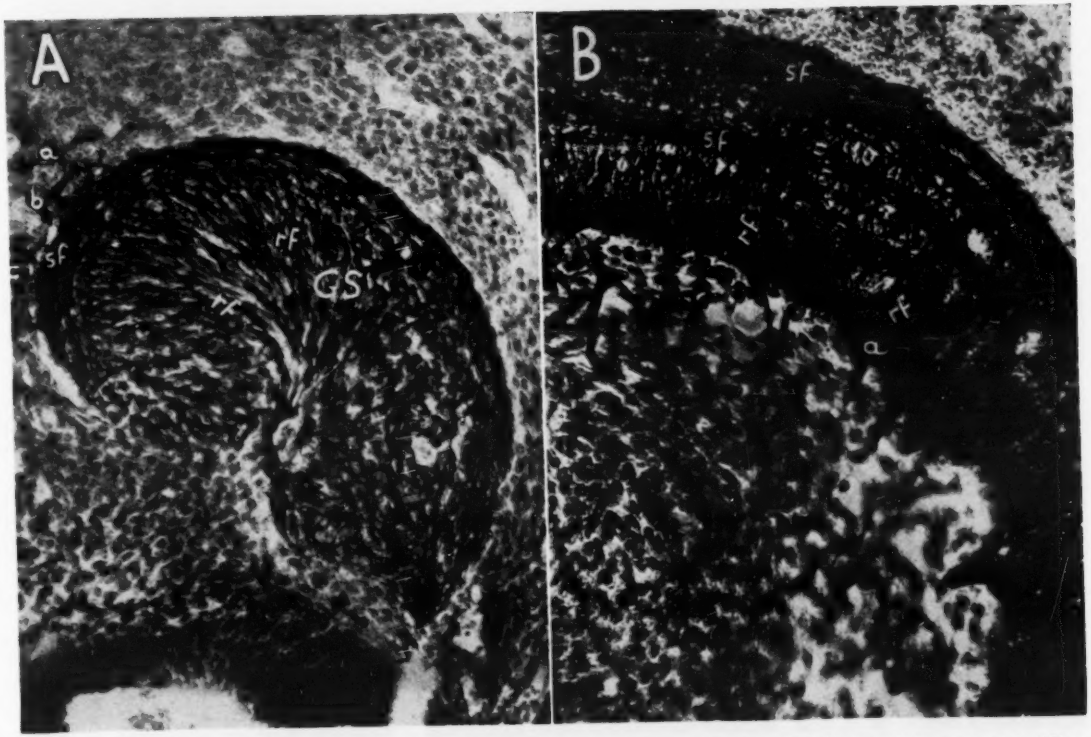

Fig. 8.—*A*, horizontal section of cochlea, apical coil. The general disposition of radial fibers, *rf*, is somewhat different from that in preceding sections, because at this level the cochlear duct extends farther than the spiral ganglion, but two systems of fibers, radial, *rf*, and spiral, *sf*, are always well evident; *a*, *b*, *c*, rami from spiral fibers spread toward connective tissue, which will form the wall of scala; *GS*, ganglion spirale (× 250).

B, horizontal section through modiolus and lamina spiralis. Spiral, *sf*, and radial, *rf*, systems of fibers are clearly shown. At *a* is a bundle of fibers entering the lamina spiralis from the modiolus, taking a spiral course immediately (× 280).

7B). If the single fibers are followed in properly orientated sections, the conviction may easily be gained that each fiber is strongly individualized and that ramifications, even if existing, must be exceptional. Briefly, the system formed by the ganglion cells and by its neurons, central and peripheral, is extraordinarily regular in its radial arrangement, and it may be safely stated that the number of the cells in the spiral ganglion corresponds strictly to the number of the central and peripheral fibers. Furthermore, the arrangement of the cells and fibers is the cause, and not the consequence, of the modality of growth of the lamina spiralis ossea with its canals, inasmuch as in our preparations this arrangement is already clearly evident while the bony lamina is not yet ossified.

In the initial portion of the cochlea, which is not yet coiled, this arrangement is apparently simpler, but substantially identical, considering the reciprocal positions of the ganglion and the cochlear duct. In sections parallel to the bases of the modiolus, a short nerve seems to run from the ganglion to the cochlear duct (Fig. 4*B*); this has been described as the nerve of the initial portion. As a matter of fact, it is nothing but a group of peripheral cochlear bundles, only one of which is entirely represented in the section, while others are only partially represented. The picture closely corresponds to that illustrated in Figure *7A*, which is observable at any level of the cochlea in sections running parallel to the axis of the modiolus. In fact, in both cases the plane of the section relative to the reciprocal positions of the ganglion and the cochlear duct is strictly similar. The arrangement of the ganglion cells and their related neurons is fundamentally the same in all cochlear coils; however, both cells and neurons tend to be bent more and more toward the apex as one moves from the base to the apex, in relation to the fact that the cochlear duct extends farther than the spiral ganglion (Fig. 8*A*).

SYSTEM OF SPIRAL FIBERS

Deiters and Kölliker had made the observation of fibers running spirally in the lamina spiralis; these fibers were interpreted as being collagenous. Boettcher [11] first gave a detailed description of such fibers—their location and arrangement—and stated that they must be considered nerve fibers. According to his description, they are grouped into clearly individualized bundles, and although their size and number vary with the animal species considered, they are always well defined in all mammals. They run parallel to the spiral ganglion on each side of it, but preferably above it, along the whole length of the cochlea, crossing the radial fibers at right angles but often entering into direct connection with them by numerous anastomoses. They end most probably by assuming sooner or later a radial course and entering the foramina nervosa, together with the spiral fibers.

The spiral fibers of Boettcher seem to have been scarcely known to some of the later investigators. Möbius [12] believed that the spiral fibers are collateral twigs of the peripheral neurons, and Ebner [13] seems to have been of a similar opinion, as he divided the peripheral neurons into *Ortoneuren* (radial, not ramified) and *Spiro-*

11. Boettcher, A.: Weitere Beiträge zur Anatomie der Schnecke, Arch. path. Anat. **17**:243, 1859.

12. Möbius, E., in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohren-Heilkunde, Berlin, Julius Springer, 1926, Pt. 6, Vol. 1, p. 34.

13. Ebner, in Kölliker, R. A.: Handbuch der Gewebelehre des Menschen, Engelmann, Leipzig, 1902, Pt. 3, Vol. 2, p. 944.

neuren (spiral, ramified). However, Kolmer [4a,b] denied the presence of ramifications or collaterals in the peripheral cochlear neurons in the lamina spiralis. Held [14] expressed the belief that the spiral bundles, far from being collaterals, are peripheral neurons themselves, running in the most different directions and often presenting a plexus-like arrangement. Lorente de Nó partially confirmed [8] the belief of Held by dividing the ganglion cells into two types: Type A, with a short straight neuron directly entering the cochlear duct, and Type B, with neurons which bend toward the apex and follow a spiral course, sending collaterals which unite with type-A neurons and enter the foramina nervosa.

The opinion of del Bo and Livan [10] is not at variance with this belief; they also described two spiral formations in the lamina spiralis, which they called the paramarginal plexus and the marginal plexus, consisting of peripheral neurons intimately intermingled and anastomosed.

In our preparations the spiral fibers may be most clearly followed in all coils (Figs. 6*A*, 6*B*, 7*A*, 8*A*, 8*B*). In the initial portion of the cochlea they are cut transversely in sections running parallel to the base of the modiolus (Fig. 5*A*) and are still well evident, although reduced in number, among the radial fibers. The aforementioned ramus of the round window seems to originate in the spiral system of fibers (Fig. 5*A*). As previously stated by Boettcher, it is remarkably difficult to define the origin of these spiral fibers.

However, in my research several facts have been established:

1. No fibers of the spiral system may ever be seen originating in the cells of the spiral ganglion or, as collaterals, from the peripheral neurons of the ganglion cells.

2. Bundles of fibers coming from the modiolus may be seen here and there entering the lamina spiralis and immediately taking a spiral course (Fig. 8*B*), running adjacent to the ganglion and joining at different levels the main spiral bundles, which are situated apically and lateral to the ganglion itself; along their entire length they cross the radial system of fibers, remaining always in a different plane (Figs. 6*A* and 9*A*).

3. It has never been possible to observe well-defined groups of spiral fibers, similar to those of the radial system, entering the foramina nervosa. On the contrary, the spiral bundles give origin to plexus-like formations in the periphery of the lamina spiralis, and isolated fibers from this plexus join the radial bundles (Fig. 9*B*). The possibility cannot be excluded that other fibers reach the walls of the scala through the lamina spiralis itself. In our preparations the process of excavation of the scala is still not completed, but fibers coming from the aforementioned plexus and running into the connective tissue which will give origin to the wall of the scala may be easily observed (Fig. 8*A*).

ARRANGEMENT OF CENTRAL COCHLEAR FIBERS

All investigators agree that the fibers of the cochlear nerve contained in the modiolus are wound in rope-like fashion, this twisting being due to the coiling of the cochlea and the related ganglion in the course of their development.

14. Held, H.: Untersuchungen über den feineren Bau des Ohrlabyrinthes der Wirbeltiere, Abhandl. math.-phys. Cl. k. sächs. Gesellsch. Wissensch. **31**:193, 1909; in Denker, A., and Kahler, O.: Handbuch der Hals-, Nasen- und Ohren-Heilkunde, Berlin, Julius Springer, 1926, Pt. 16, Vol. 1, p. 340.

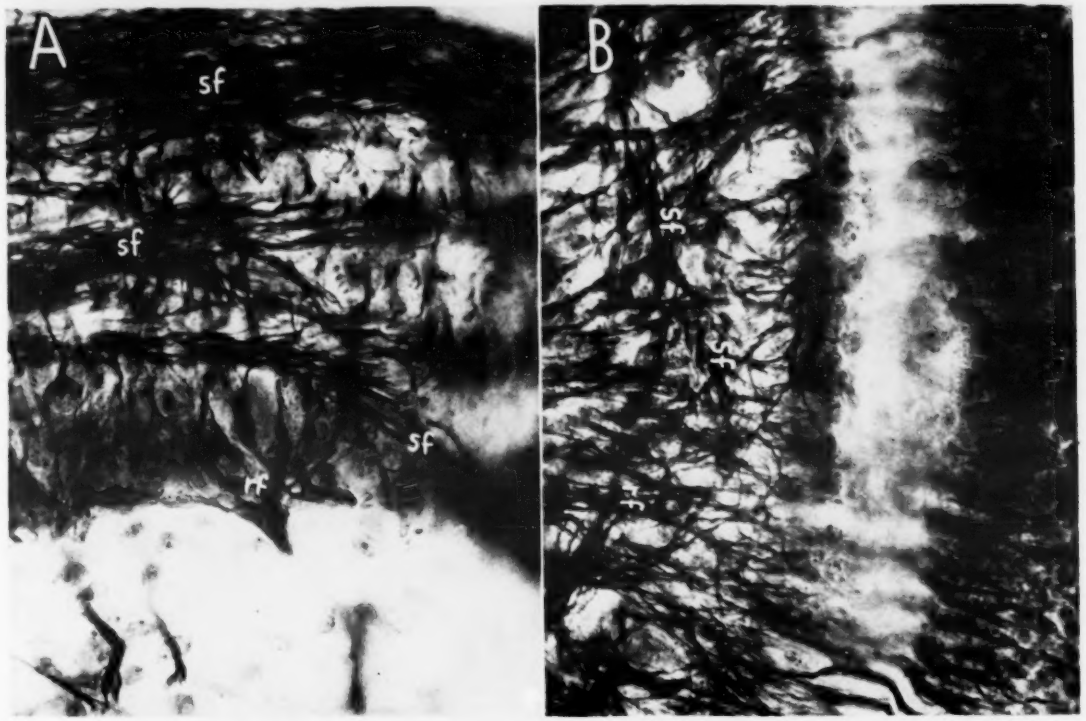

Fig. 9.—*A*, horizontal section through lamina spiralis, middle coil. The spiral system of fibers, *sf*, is focused, while the radial system, *rf*, is poorly visible in the background. From a comparison with Figure 6*A*, representing the same point, it is clear that radial and spiral fibers are in two different planes, and run independent of each other (× 630).

B, horizontal section through the lamina spiralis, middle coil. At periphery, spiral fibers form a plexus, from which some twigs join the radial fibers entering the foramina nervosa: *rf*, radial fibers; *sf*, spiral fibers (× 700).

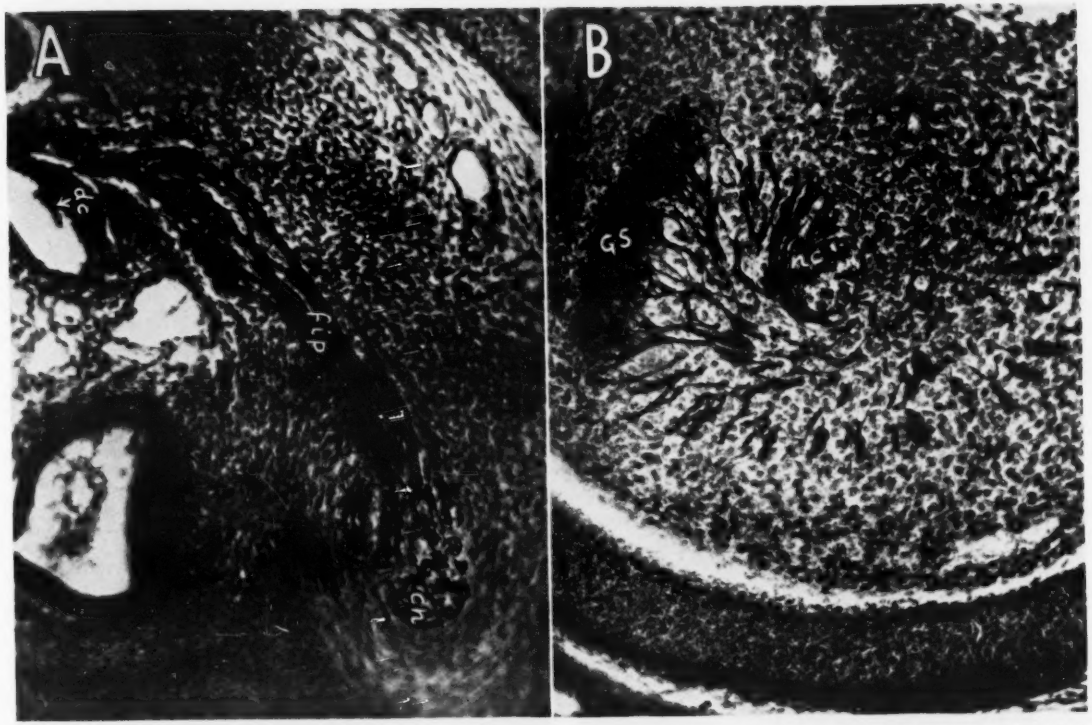

Fig. 10.—*A*, horizontal section through the base of the modiolus. The fiber coming from the initial portion, *fip*, of the cochlea runs straight into the modiolus; *dc*, ductus cochlearis; *cn*, cochlear nerve (× 135).

B, horizontal section of cochlea, middle coil. The winding of the fibers coming from ganglion, shown partially in *GS*, is well evident; *dc*, ductus cochlearis (× 175).

Scant attention, however, has been paid to the modality of this twisting by former investigators, and only Poljak [15] and Lorente de Nó [7a] have given a more detailed description of it. According to their findings, the fibers coming from that part of the ganglion which is situated at the distance of a half-turn from the round window run straight into the modiolus and form the axis of the nerve. The fibers coming from the remaining sections of the ganglion wind spirally around this axis, those coming from the apex in one direction and those coming from the base in the opposite direction.

Fig. 11.—Horizontal section of cochlea; middle coil. Anastomosis between central fibers outside ganglion (a, a,) are clearly visible; GS, spiral ganglion ($\times$ 320).

Bolk, Göppert, and associates [16] on the contrary, stated that all the fibers wind in one direction only. According to Poljak,[17] Lewy and Kobrak,[18] and Lorente

15. Poljak, S.: The Connections of the Acoustic Nerve, J. Anat. **60**:465, 1926.

16. Bolk, L.; Göppert, E.; Kallius, E., and Lubósch, W.: Handbuch der vergleichenden Anatomie der Wirbeltiere, Berlin, Urban & Schwarzenberg, 1934, Vol. 2, Pt. 2, p. 1360.

17. Poljak, S.: Über die neusten Untersuchungen am Oktavus-System der Säugetiere, Zentralbl, Hals-, Nasen- und Ohrenh. **11**:98, 1928; Über den allgemeinen Bauplan des Gehörsystems, Ztschr. ges. Neurol. u. Psychiat. **110**:1, 1927.

18. Lewy, F. H., and Kobrak, H.: The Neural Projection of the Cochlear Spirals on the Primary Acoustic Centers, Arch. Neurol. & Psychiat. **35**:839 (April) 1936.

de Nó,[7a] it is possible to a certain extent to follow the single fibers from the spiral ganglion along the modiolus down to the cochlear nuclei, thus demonstrating that each fiber keeps its strict individuality, and giving support to the theories regarding the cochlea as a tone analyzer. However, von Bardeleben [19] reported the existence of many anastomoses among the cochlear fibers between their exit from the ganglion and their entrance into the modiolus.

My observations on these sections allowed me to define the arrangement of the cochlear fibers in the modiolus. Thus, the fibers proceeding from the initial portion of the ganglion run unwound into the modiolus, being first situated eccentrically to the trunk of the nerve and moving gradually to occupy approximately the center of it. All other fibers coming from the coiled part of the ganglion show a spiral winding, which is always in the same direction and corresponds to the direction of the cochlear coils in respect to the modiolus (Fig. 10*A*, *B*). The fibers coming from the apex occupy the central part of the nerve, around the unwound fibers of the initial portion, and the fibers coming from the middle and basal coils arrange themselves more and more peripherally as they proceed from the middle to the basal coil. I have been able to determine that each fiber remains well individualized in its course from the ganglion into the modiolus; however, it anastomoses rather freely just outside the ganglion, between it and the modiolus (Fig. 11).

SUMMARY AND CONCLUSIONS

The inner ear of rat fetuses at different stages of development has been thoroughly examined in serial sections of the head, stained according to the method of Cajal, with the aim of contributing to a better knowledge of the innervation of the cochlea. The existence of vestibulocochlear anastomosis has been confirmed. Furthermore, the existence of two systems of fibers in the lamina spiralis has been clearly demonstrated, one of which is radially arranged and has its origin in the cells of the spiral ganglion, while the other is spirally arranged and is independent of the ganglion itself.

It is presumable that the spiral system derives from the vesibular ganglion, and that it distributes to the walls of the cochlear duct, somehow integrating the strictly specific sensorial function of the radial system.

From the sum of these observations it is beyond doubt that two systems of fibers provide the innervation of the cochlea, one of which is radially, and the other spirally, arranged.

The strict dependence of the radial system on the spiral ganglion cannot be discussed: This system is characterized by the great regularity of its disposition, with regard both to the central and the peripheral neurons and to the ganglion cells. The destination of this system of fibers to the innervation of the organ of Corti, likewise, cannot be discussed.

Far less certain is the interpretation of the significance of the spiral system. Inasmuch as a direct continuity of its fibers with the cells of the spiral ganglion has been always impossible to demonstrate, the question arises whether one is

19. von Bardeleben, K.: Handbuch der Anatomie des Menschen, Jena, Fischer, 1905, p. 315.

dealing with vegetative or with sensory fibers.[20] Although the possibility cannot be excluded that some of the fibers of this system may be vegetative in character, it is certain that they must, even if existing, be extremely scanty, since this system is almost entirely formed of large myelinized fibers, having the same appearance as the fibers of the radial system, in sections treated with the common stains. Their staining properties, even more than any other consideration, rule out the possibility of their belonging to the autonomic nervous system. If these findings are related to the equally undoubted observation of a large number of peripheral fibers of vestibular origin entering the cochlear nerve,[21] it becomes logical to suppose that the origin of the spiral fibers may be the vestibular ganglion, i. e., its modiolar extension. A demonstration of such a hypothesis is attainable only experimentally, and experiments along this line have been started.

As to the peripheral termination of the spiral fibers, I have not been able to collect much information; anyway, it seems sufficiently clear that they give origin in the lamina spiralis to a plexus whose ramifications seem to penetrate somehow into the cochlear canal. In this regard it may be useful to recall the observations of Palumbi,[9] who demonstrated in all parts of the human labyrinth a system of nonspecific sensory innervation alongside the system of specific sensorial innervation. In case the derivation of the spiral system from the vestibular ganglion could be demonstrated, the sensory apparatus of Palumbi might also be thought to derive from the same ganglion or its extensions.

The demonstration afforded of two systems of fibers innervating the cochlea, dissimilar in arrangement, course, and probably in origin, raises again the problem of a dual cochlear innervation. In 1902 Hensen,[22] considering the physiology of the auditory apparatus, argued the possible existence of two sensorial apparatuses in the cochlea, for tone and noise perception, respectively. The problem was again treated by Jaensch,[23] who stated that the auditory organ, like the eye, must possess special senses differing in their differentiation and phylogenetic age, such as to make possible not only the perception of single tones but also the appreciation of the quality and volume of sounds. Roenau [24] discussed an aspect of the same problem. Comparative clinical and histopathological studies by Saxen [25] led to the conclusion that good hearing for the voice may be preserved in the presence of severe degeneration of the organ of Corti. The ear may therefore hear without the organ of Corti, provided that the ganglion and the epithelial and secretory structures of

20. Bovero, A.: Connessioni simpatiche del ganglio vestibolare del nervo acustico, Arch. ital. otol. **25**:41, 1914. Udvarhely, K.: Vestibulare Nerven-Verbindungen, Ztschr. Ohrenh. **67**:136, 1913.

21. Rasmussen, A. T.: Studies of the Eighth Cranial Nerve in Man, Laryngoscope **50**:67, 1940.

22. Hensen, V.: Fortschritte in einigen Teilen der Physiologie des Gehörs, Ergebn. Physiol. **1**:2, 1902.

23. Jaensch, E. R.: Untersuchungen über Grundfragen der Akustik und Tonpsychologie, Leipzig, J. A. Barth, 1929.

24. Roenau, E.: Zur Psychologie des Hörens, Monatsschr. Ohrenh. **71**:728, 1937.

25. Saxen, A.: Pathologie und Klinik der Alterschwerhörigkeit, Acta otolaryn., Supp. 23, p. 1, 1937.

the cochlear canal remain undamaged. Kobrak [26] was prompted by these observations to reconsider the question, reaching the conclusion that the human ear may be formed by two phylogenetically different sensorial systems, to one of which, the younger and more differentiated, or "cochlearis proper" principle, should be ascribed the frequency analysis, while to the other, older and less differentiated, or "cochleovestibular principle," should be ascribed the less specific function of the perception of the fused tonal sensation and of the volume—in one word, of the quasitactile, "spatial" character of the sound. However, no anatomic or experimental data have so far been available to lend morphologic support to these theories. The demonstration afforded by the present research of two systems of fibers in the cochlea possibly independent of each other throws new light upon the problem and gives new impulse to research along this line. It is certain that only a successful experimental demonstration of the kind now being tried could bring a decisive contribution to the solution of the fascinating problem of the dual sensory innervation of the cochlea.

University of Milan.

26. Kobrak, F.: Betrachtungen zur Arbeitshypothese eines cochleo-vestibularen Hörprinzips, Pract. oto-rhino-laryng. **9**:346, 1947; On the Nature of a Dual Function in the Cochlear Nerve, J. Laryng. & Otol. **63**:7, 1949.

Case Reports

SUPERNUMERARY MUSCLE OF MIDDLE EAR

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SUPERNUMERARY muscles of the middle ear have seldom been observed on examination of histologic sections of the temporal bone. A thorough review of our collection of over 350 temporal bones cut serially disclosed the presence of such a muscle in only one instance. It was felt, therefore, that a report of this case was worth while. A brief review of the literature revealed that Schwabach [1] in 1907 was the first to report a case in which an anomalous muscle of the middle ear was found. The patient was a deaf-mute of 44 who died of carcinoma of the ventricle. The small abnormal muscle, observed microscopically, was situated within the Fallopian canal near the knee of the facial nerve and was in no way connected with the tensor tympani or the stapedius muscle. Schwabach attempted to establish a causal relationship between this muscle and the deaf-mutism in his case. In 1930 Brock [2] reviewed a microscopic series of 277 temporal bones and could find five cases with supernumerary muscles in the region of the facial nerve. In four of these the muscle lay within the Fallopian canal and in this respect resembled very closely the case previously described by Schwabach. In the fifth case the muscle occupied a bony canal of its own and was separated from the facial nerve by a thin layer of bone. The nerve supply to this muscle could not be determined in any of the five cases in spite of careful search, nor could a satisfactory explanation be offered with regard to the derivation of the muscle. Also, in none of the five cases were there present any other anomalous conditions of the bony labyrinth or middle ear. Of significance was the observation, made clinically, that in none of the cases was there a hearing defect which could be attributed to this muscle. Brock concluded from these findings that the supernumerary muscle, when found associated with a hearing impairment, is in all probability not the causative factor responsible for the hearing impairment.

The occurrence of supernumerary muscles in the presence of other congenital anomalies of the middle ear must also be considered very rare. Marx [3] in his classic and comprehensive article on congenital malformations of the ear makes no mention of it. However, Altmann,[4] who more recently reviewed a very large series

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1. Schwabach: Beitrag zur Anatomie der Taubstummheit, in Denker, A., and Bergmann, J. F.: Die Anatomie der Taubstummheit, Deutschen Otologischen Gesellschaft, Weisbaden, 1907, p. 21.

2. Brock, W.: Über abnorme Muskelbildung im beziehungsweise am Fazialiskanal, Monatsschr. Ohrenh. **64**:933, 1930.

3. Marx, H.: Die Missbildungen des Ohres, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, Vol. 12, p. 609.

4. Altmann, F.: Zur Kenntnis der überzähligen Mittelohrmuskeln, Arch. Ohren-, Nasen- u. Kehlkopfh. **133**:28, 1932.

of such cases, could find only three instances with supernumerary muscles; these three cases he reported in great detail. He stated that while the absence of either one or two muscles of the middle ear is not a rare occurrence, particularly when associated with other middle ear anomalies and especially with anomalies of the auditory ossicles, the presence of supernumerary muscles in such instances is indeed a very rare one. Altmann was able to determine from his microscopic section the nerve supply of the anomalous muscle in each of the three instances reported by him: the trigeminal or the facial nerve. Of interest, was the finding in one of his cases of two supernumerary muscles. From comparative anatomical studies he was led to believe that this muscle is probably a "rest" of the depressor mandibulae in nonmammals, which functions to open the primary jaw joint, this joint in the phylogenetic development becoming in mammals the incudomalleal joint.

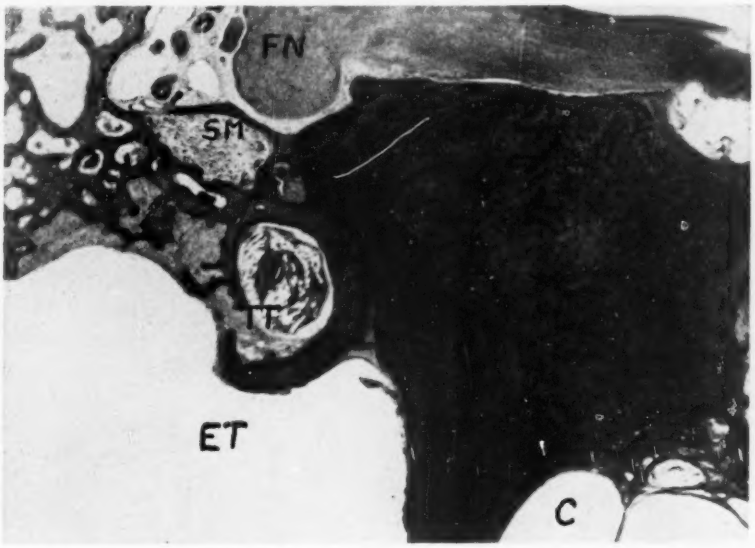

Fig. 1.—Section through the Eustachian tube showing the supernumerary muscle (*SM*) and its relationship to the facial nerve (*FN*) and the tensor tympani muscle (*TT*). *C* represents the cochlea and *ET* the Eustachian tube.

REPORT OF A CASE

History.—A white man, H. R., aged 64, was admitted to the urological service because of acute urinary retention and angina pectoris. The interesting feature in the history from the otologic standpoint was that the patient had been partially deaf for over 40 years following an explosion of a shell near him. Unfortunately, a complete otologic work-up, including cochlear and vestibular tests, was not made. The patient died of coronary occlusion six days after his admission to the hospital, and the right temporal bone was procured for histologic study.

Histological Observation of the Right Temporal Bone.—Serial sections were cut vertical to the long axis of the petrosa. On the medial surface of the tympanum, at the level of its junction with the Eustachian tube and situated immediately above the canal for the tensor tympani muscle and somewhat anteroinferiorly to the facial nerve, there was present a small abnormal muscle (Fig. 1). This muscle was spindle-shaped and measured 2.5 mm. in length and 0.75 mm. in width. It was entirely contained within its own bony canal and was separated

from the facial nerve by a thin layer of bone. It did not communicate with the tensor tympani. The individual muscle fibers, cut at right angles, were striated and appeared to be normal in every respect (Fig. 2). There was no evidence of atrophy. They were surrounded by connective tissue with a rich blood supply and with many small nerve bundles. The source of the nerve supply to the muscle could not be determined with any degree of certainty in spite of careful search. There were no deposits of fat (such as are seen in the tensor tympani) within the canal. The tensor tympani and stapedius muscles showed no abnormalities with regard to morphologic structure, course, or insertion. No other anomalies were observed in the temporal bone, with the possible exception of the presence of two unusually large fossa subarcuata vessels, which in their course through the petrosa gradually merged and emptied into the superior petrosal vein. Within the tympanum fibrous connective tissue bands were seen to extend from the ossicles to the inner bony wall. The tympanic membrane was intact and showed no abnormalities.

Two distinct otosclerotic areas were present in the labyrinthine capsule. One, situated at the usual site anterior to the oval window, appeared to be of long standing and was found to

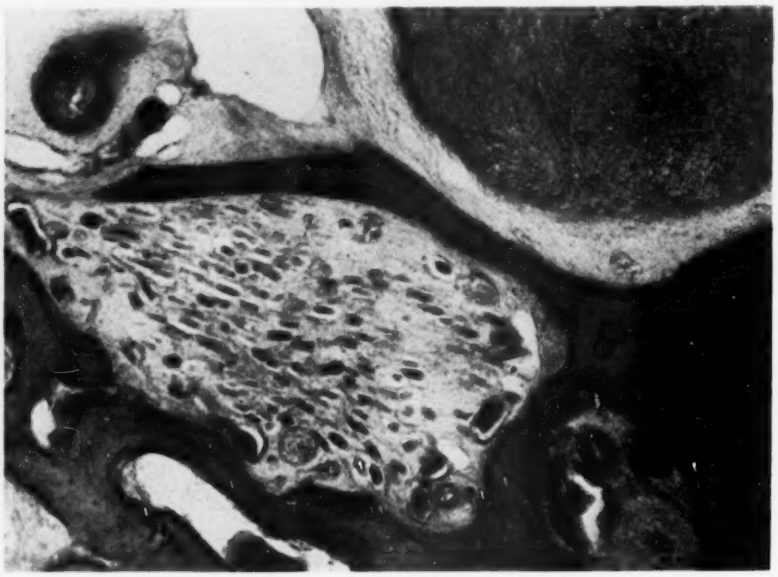

Fig. 2.—Higher magnification of Figure 1 showing the individual striated muscle fibers, nerve filaments, and dilated capillaries within the supernumerary muscle.

extend into the oval window, resulting in ankylosis of the anterior crus of the stapes; the other otosclerotic focus was situated at the apex of the cochlea and appeared to be of more recent origin than the first.

Atrophic degenerative changes were seen in the organ of Corti, in the nerve fibers within the lamina spiralis ossea, and in the cells of the spiral ganglion. This was particularly true for the first turn of the cochlea. That some of these changes were agonal in nature was considered as a possibility.

COMMENT

The anomalous muscle of the middle ear in this case was found histologically to be entirely confined to its own canal and apparently had no tendinous attachments elsewhere. Although scattered nerve filaments were seen within the muscle proper, its exact nerve supply could not be definitely determined from a study of all the sections. Any attempt to assign a definitive physiological function to this muscle

in these circumstances, therefore, would be purely speculative. Of interest is the observation that, in spite of complete lack of evidence of any functional activity of the muscle, the individual muscle fibers morphologically appeared entirely normal and showed no signs of atrophy. This observation was also made independently by Brock and by Altmann from a study of their cases. In this connection it is worth noting that in a congenitally malformed ear reported by Kelemen [5] the stapedius muscle, although appearing normal in size and structure, contained no traces of a tendon, yet the individual muscle fibers also showed no signs whatever of atrophy. Although much information has been obtained from anatomical dissections [6] and experimental work done on living animals and on living human subjects [7] regarding the physiological action of the muscles in the region of the Eustachian tube, complete agreement among otologists as a result of these investigations has not yet been arrived at. Zuckerkandl [8] in 1884 pointed out that, in general, a portion of the tensor tympani does not become tendinous and does not insert into the hammer handle, thus implying a possible secondary function of the muscle. Spiesman,[9] who made a study of the fat content in the tensor tympani in a large series of cases, found that the proportion of fat varied from the normal under certain pathological conditions of the middle ear in which this muscle was involved. Since he observed a relatively normal amount of fat in the tensor tympani of congenitally deaf ears in which during life there could be no question as to functional activity, he inferred from this that the muscle, in all probability, normally possessed an additional function to that of the acoustic.

The histologic finding in the present case of otosclerotic foci in the labyrinthine capsule with ankylosis of the stapes was in all probability purely coincidental. Nevertheless it was a most unexpected finding, particularly in view of the clinical history of deafness, the onset of which had followed a shell explosion many years before. From our knowledge of the subject a causal relationship between the trauma to the ear and the pathologic finding of otosclerosis can hardly be reconciled. The atrophic degenerative changes in the end-organ of the cochlea, however, can be ascribed to the trauma, although it is well known that these changes are also not infrequently seen in the late stages of otosclerosis.

SUMMARY

A case of supernumerary muscle of the middle ear is reported. A brief review of the literature on this subject, including a discussion of some of the theories with regard to the genesis, morphologic development, and physiological function of this anomalous muscle in general, is presented.

5. Kelemen, G.: Malformation Involving External, Middle and Internal Ear, with Otosclerotic Focus, Arch. Otolaryng. **37**:183 (Feb.) 1943.

6. Kobrak, H.: The Inner Ear: A Cinematographic Study of Some Aspects of Its Morphology, Physiology, and Pathology, Presented at the Meeting of the American Otological Society, 1951.

7. Perlman, H.: Mouth of the Eustachian Tube: Action During Swallowing and Phonation, A. M. A. Arch. Otolaryng. **53**:353 (April) 1951.

8. Zuckerkandl, E.: Zur Morphologie des Musculus tensor tympani, Arch. Ohren. **20**:104, 1884.

9. Spiesman, I. G.: Über die Fetteinlagerungen im Musculus tensor tympani am gesunden und am kranken Ohr nebst entwicklungsgeschichtlichen und vergleichend-anatomischen Bemerkungen, Monatsschr. Ohrenh. **61**:971, 1927.

CYLINDROMA IN NOSE AND SINUSES

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THE HISTOLOGIC entity most commonly called cylindroma, but known also by a variety of other names (adenoid cystic basal cell carcinoma, adenocarcinoma of cylindroma type, adenomyoepithelioma, and cellular mixed tumor) is encountered in many branches of surgery. The so-called cylindroma occurs wherever salivary or mucous glands are found. Despite its ubiquitous nature, however, confusion exists as to its proper genesis, classification, and clinical picture.

Billroth [1] first used the term cylindroma in 1856 to describe a tumor which had arisen from the mucous glands of the accessory nasal sinuses, and penetrated into the orbit.[2] He referred to a tumor composed of glossy translucent cylinders of hyaline connective tissue between which ran a network of tumor cells.[3] Since then, similar tumors have been frequently recognized in the oral cavity, nasopharynx, and salivary glands, as well as in the skin, larynx, and bronchi.

The lack of unanimity of opinion regarding the nature of this tumor is adversely reflected in the results of surgical treatment.[4] Sometimes it is called simply a "mixed tumor" and given a benign classification, while at other times it is termed "potentially malignant."

Bauer and Fox [2] state that the tumor though genuinely benign may undergo cancerous alteration. McFarland [5] emphasized the lack of correlation between the histopathology of the tumor and its clinical behavior. Dockerty and Mayo [4] first classified it with the mixed tumors and were impressed by the extremely high rate of recurrence following conservative operation. In a later study [6] they reported that eight of 20 traced patients with this type of tumor died of metastases. Ackerman and del Regato [7] classified it with the semimalignant tumors but noted that it is

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1. Billroth, T.: Beobachtungen über Geschwülste der Speicheldrüsen, Virchows Arch. path. Anat. **17**:357 (Sept.) 1859.

2. Bauer, W. H., and Fox, R. A.: Adenomyoepithelioma (Cylindroma) of Palatal Mucous Glands, Arch. Path. **39**:96 (Feb.) 1945.

3. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, Philadelphia, W. B. Saunders Company, 1940, p. 339.

4. Dockerty, M. B., and Mayo, C. W.: Primary Tumors of the Submaxillary Gland with Special Reference to Mixed Tumors, Surg., Gynec. & Obst. **74**:1033 (June) 1942.

5. McFarland, J.: Three Hundred Mixed Tumors of the Salivary Glands, of Which 69 Recurred, Surg., Gynec. & Obst. **63**:457 (Oct.) 1936.

6. Dockerty, M. B., and Mayo, C. W.: "Cylindroma" (Adenocarcinoma, Cylindroma Type): Report of 2 Cases with Metastasis, Surgery **13**:416 (March) 1943.

7. Ackerman, L. V., and del Regato, J. A.: Cancer: Diagnosis, Treatment, and Prognosis, St. Louis, C. V. Mosby Company, 1947, p. 632.

much more malignant than is generally suspected. Quattlebaum and co-workers [8] pointed out its very malignant nature and suggested that treatment include block node dissection when feasible. They also pointed out that the pathologist familiar with the frozen section appearance of this tumor can render a real service to his surgical colleagues.

Unfortunately, the insidious malignant nature of this tumor is not recognized by many clinicians.[9] Some authors do not accept cylindroma as a cancer.[2] The following case reports, of which the first two are reported in detail, will serve to emphasize the clinical behavior and characteristics of this tumor.

REPORT OF CASES

CASE 1.—R. C., a 33-year-old, white, American-born housewife, noted the onset of numbness over the left cheek five years before her first visit, on Dec. 30, 1949. As this symptom slowly progressed, she began to have difficulty in opening her mouth. Thorough neurological investigation yielded no obvious cause for her symptoms. Several upper and lower teeth were extracted, without relief. Roentgenograms of the sinuses at that time showed only a thickened membrane of the left antrum.

Two years later she first described pain beneath and behind the left orbit, with occasional attacks of blurred vision in the left eye. Trismus increased, and she began to notice a slight serosanguineous discharge from the left nares. She began to have pain in front and behind the left ear. Sinus roentgenograms were repeated in October, 1949, five years after the onset of her first symptoms. They revealed destruction of the posterior, medial, and superior walls of the left antrum. A biopsy specimen was taken, which revealed adenocarcinoma, basal glandular pattern (cylindroma).

When the patient was first seen, on Dec. 30, 1949, physical examination revealed slight proptosis of the left eye. The left pupil was dilated more than the right, but extraocular movements were unremarkable. The skin over the left cheek was dull to touch and showed slight redness and fullness. There were induration and swelling in the preauricular area. Trismus was moderately advanced, the space between the central incisor teeth measuring 1.5 cm. at maximum extension. Mirror examination of the nasopharynx revealed a 2-cm., smooth bulge along the left lateral wall partially blocking the orifice of the Eustachian tube. There were no significantly enlarged neck nodes. Eye consultation revealed normal fundi. Chest roentgenograms showed no metastasis.

On Jan. 13, 1950, a bilateral external carotid ligation and total resection of the left maxilla were performed. A cheek-flap approach was used by splitting the upper lip in the mid-line, extending the incision paranasally and infraorbitally, and retracting the cheek laterally to expose the entire underlying maxilla.

The operative findings revealed a very extensive, infiltrative tumor (Fig. 1) which invaded the pterygomaxillary space, the capsule of the temporomandibular joint, and all walls of the maxillary sinus. The tumor had eroded the floor of the orbit and extended medially and superiorly into the anterior fossa of the skull.

The maxillary and ethmoid sinuses were removed, the pterygoid muscles were resected, the coronoid process of the mandible and the posterior pterygoid plate divided and removed along with the capsule of the temporomandibular joint, which gave marked relief of trismus. Because of the apparent hopelessness of the condition, and because of the extent of cancer within the orbit, an exenteration was not performed. A split-thickness skin graft was placed over the operative defect, lining the raw inner surface of the cheek and the floor of the orbit as well. The wound edges were approximated. Postoperative convalescence was uneventful, and a dental prosthesis was made within four weeks to fill the defect in the hard palate on the resected side.

8. Quattlebaum, F. W.; Dockerty, M. B., and Mayo, C. W.: Adenocarcinoma, Cylindroma Type, of Parotid Gland: Clinical and Pathologic Study of 21 Cases, Surg., Gynec. & Obst. **82**:342 (March) 1946.

9. Personal communications to the author.

She was given 25 x-ray treatments in 34 days after complete healing had taken place. The dosage was as follows: 400 kv. with a half-value layer of 4.5 mm. Cu; 3,075 r in air to a left anterior facial field measuring 4.5 by 5 cm.; 2,900 r in air to a left lateral facial field measuring 8 by 8 cm.; calculated tumor dose at 4 cm. was 5,061 r.

The patient enjoyed good health with no morbidity and excellent palliation until January, 1951, exactly one year after operation. At this time the left eye, which had given her no

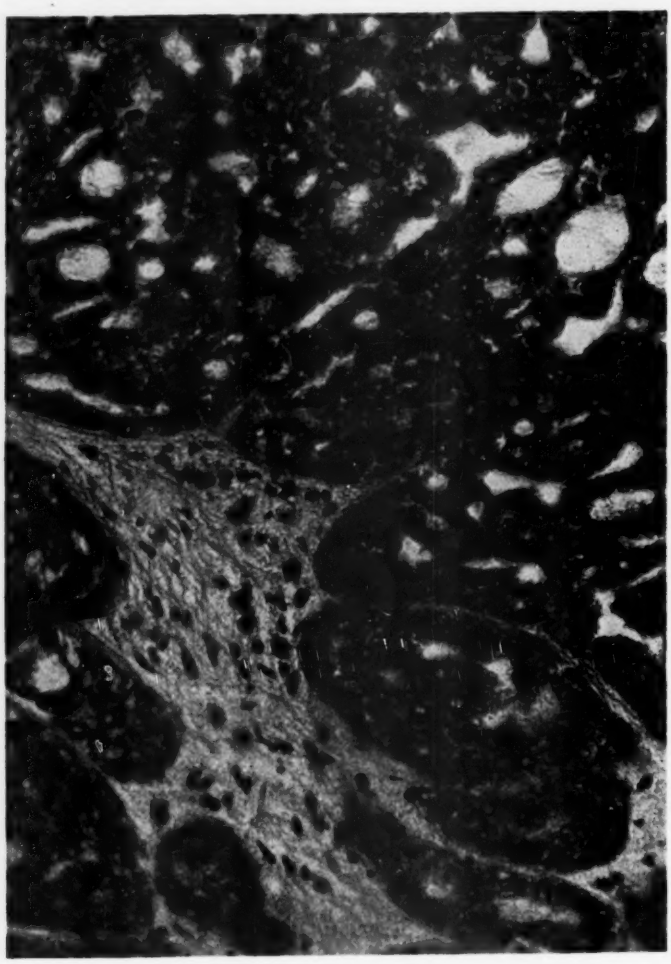

Fig. 1.—Typical structure of adenoid cystic basal cell carcinoma from tumor removed in Case 1.

trouble postoperatively, suddenly became markedly proptosed. There was still no gross evidence of residual cancer within the operative site, and a chest roentgenogram at this time showed no metastasis. On Jan. 11, an exenteration of the orbital contents was performed, and postoperatively she received the following x-ray treatments to the orbital area: 200 kv. with a half-value layer of 1 mm. Cu; 50 cm. target-skin distance; 200 r daily, alternated through a 10 by 10 cm. lateral port and an 8 by 6 cm. anterior port for a total of 1,900 r and 2,000 r (air)

respectively. She remained well until March, 1951, at which time she began to complain of severe pain in the cervical spine. Roentgenograms of the spine showed no metastasis, but the lungs revealed multiple small densities (Fig. 2) which can be interpreted only as metastatic cancer. The course since then has been progressively downhill, so that at the present time (August, 1951) her death is imminent from widespread cancer metastasis to both skeleton and soft tissues.

CASE 2.—E. H., a 26-year-old, white, American-born woman, was first seen on Feb. 24, 1951. In 1944 and again in 1947 she had two attacks of hemoptysis, but roentgenograms revealed no lung lesions, and she enjoyed good health until December, 1950, when she noticed a slightly sanguineous discharge from the left nasal passage. This was followed by numbness and dullness over the left cheek, blurred vision in the left eye, and difficulty in moving the eye laterally. There was also difficulty in opening her mouth. Despite numerous examinations by several physicians, there was no diagnosis until February, 1951, when a biopsy was taken of a small protruding tumor of the left nasal passage. The report was adenoid cystic basal cell carcinoma (cylindroma). Roentgenograms of the sinuses which were taken at that time showed a well-outlined large tumor of the left antrum, with erosion of the medial wall and extension through

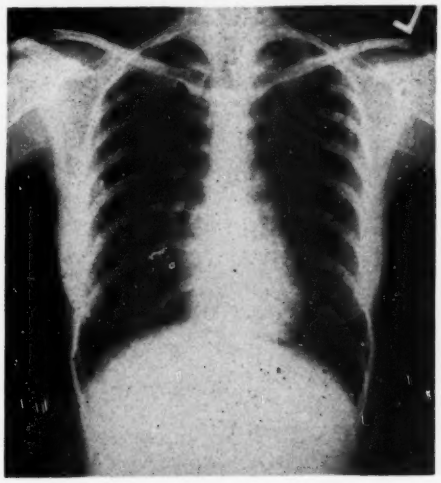

Fig. 2.—Multiple small densities in lung (Case 1).

the floor of the orbit with destruction of the wing of the sphenoid in the region of the foramen ovale. Chest roentgenograms revealed multiple nodular parenchymal densities in both lung fields, with moderate pleural effusion bilaterally.

Physical examination revealed a chronically ill, weak, pale young woman who was in no pain but was extremely apprehensive. There was no swelling of the left cheek, and the left eye appeared unremarkable, but there was definite impairment of left lateral rectus function. On the left side of the hard palate there was a 1-cm., superficial, tender ulcer which bled easily on contact. The left nasal passage revealed no tumor, and mirror examination of the nasopharynx was unremarkable. A 2-cm., slightly tender lymph node was felt in the upper jugular chain just behind the angle of the mandible on the left side. There were no malignant cells found in an examination of 500 cc. of straw-colored fluid removed from the right pleural cavity. The impression was advanced cancer of the left maxillary sinus with lung metastases and intracranial involvement. Palliative irradiation therapy was recommended.

The patient was given the following x-ray dosage: Anterior and lateral ports were used to include the left orbit and antrum; the lateral port measured 11 by 7 cm. and the anterior 6 by 8 cm. She was given 200 r (air) to one port daily from March 1 to March 12, 1951. The total

air dose to each port was 3,400 r, and the minimum tumor dose was 3,500 r. Other factors were the following: 200 kvp., 0.33 mm. Cu, 1 mm. Al filtration, target-skin distance 50 cm., and half-value layer 0.95 mm. Cu.

An amazing clinical improvement followed this course of treatment, so much so that doubt was cast on the original diagnosis. In the ensuing weeks, the node in the left of the neck disappeared, complete eye function returned, and the lung involvement, instead of progressing, remained unchanged. The possibility of a concomitant inflammatory chest disease was entertained, especially in view of a past history of hemoptysis. Sinus roentgenograms showed marked reduction in the size of the tumor of the left antrum.

Because of these factors, irradiation was carried to a cancericidal dosage, and maxillary resection was performed on April 30, 1951. The operation was modified, however, because of the uncertainty of the pulmonary condition. Through an upper lip splitting, paranasal and infraorbital incision the cheek flap was laid back laterally, exposing the entire underlying maxilla. The floor (hard palate) of the antrum was not disturbed, but the entire anterior and lateral walls were removed, revealing an oval bony mass 3.5 by 2.4 by 2.5 cm., with a smooth,

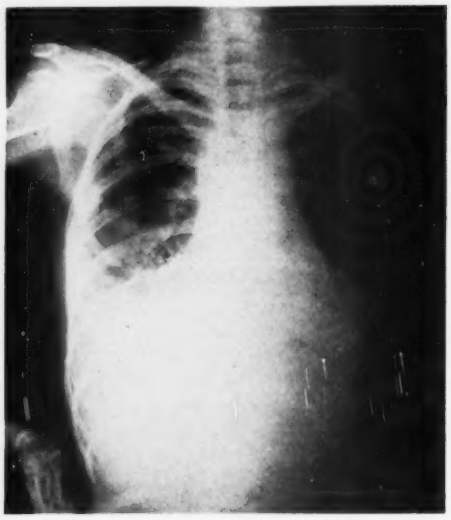

Fig. 3.—Parenchymal infiltration with mediastinal widening (Case 2).

glistening yellow surface, situated within the antrum and eroding the floor of the orbit. There was no other evidence of tumor. Frozen sections of the lining of the antrum and periorbital structures showed no cancer. The bony tumor was easily removed, the sinus loosely packed so as to support the fragmented orbital floor, and the cheek flap closed. The packing was removed on the eighth postoperative day after an uneventful postoperative course. The pathologist's report was "osteoma, maxillary sinus; sinusitis, chronic, mild."

One month after discharge, however, the patient became markedly dyspneic. She began to have an uncontrolled, nonproductive cough. Roentgenograms of the chest taken on June 12 revealed for the first time mediastinal widening (Fig. 3) but no increased parenchymal infiltration. Because no cancer had been found on maxillary resection, lung metastasis was no longer considered as a likely diagnosis. There was a rapidly downhill course, nevertheless, and she died on June 23, four months after her first visit. Autopsy revealed extensive infiltration with cancer of both lungs, pleura, myocardium, liver (Fig. 4) peritoneal surfaces, uterus, and ovaries. In addition, special postmortem studies of the left orbital and maxillary regions revealed small, focal areas of adenoid cystic basal cell carcinoma identical with that obtained on biopsy four months previously.

CASE 3.—W. H., a 29-year-old, white, American-born farmer, was first seen in the office of Dr. R. C. McNaught on Sept. 14, 1950, complaining of obstruction of the left nostril and a feeling of pressure in his left ear for one year. Previous treatments had consisted in attempts at inflation of the left Eustachian tube and systemic administration of penicillin until a biopsy of a large granular tumor obstructing the left posterior choanae and the orifice to the Eustachian tube was done. It was reported as a nonkeratinizing type of epidermoid carcinoma, but on review of the slide here was called a cylindromatous type of adenocarcinoma. X-ray treatment to the nasopharynx was started, but the tumor showed no response after 1,000 r had been given. On Oct. 19, 1950, Dr. McNaught removed the tumor through a radical antrotomy, with excision of the posterior soft and hard palates; the posterior ethmoid and sphenoid sinuses were opened and showed no cancer. A wide area was thus made accessible, through which radium was applied. An exposure of 2,625 mg.-hr. was estimated to have delivered a minimum tumor dose of 5,000 gamma r in 10 days. The patient did well until July, 1951, which was 10 months after operation; at this time cough and dyspnea developed. Chest roentgenograms revealed metastases, and at the present time (August, 1951) the patient is dying of cancer.

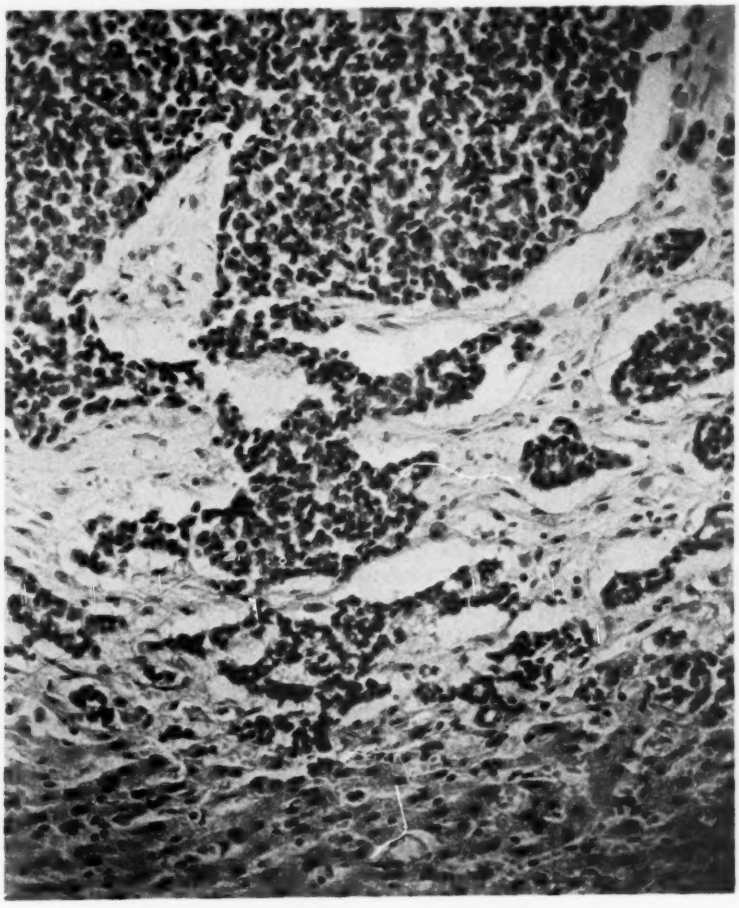

Fig. 4.—Liver metastasis present in Case 2.

CASE 4.—C. H., a 41-year-old, white, American-born woman, was first seen in the office of Dr. R. C. McNaught on Oct. 3, 1945, complaining of left nasal obstruction found to be due to allergic swelling. Thorough examination at that time revealed no evidence of tumor, but in January, 1947 (15 months later), a polypoid tumor was seen in the left naris and biopsy revealed a cylindroma. Symptoms at that time included occasional attacks of epistaxis, numbness over the left infraorbital ridge, some slight protrusion of the left eye, and blurring of vision in this eye on looking to the extreme right. An x-ray tumor dose of about 6,000 r was delivered to the tumor, with no response and actual increase in size. On May 7, 1947, Dr. McNaught performed a left external ethmosphenoidectomy and left radical antrotomy and inserted 100 mg. of radium into the posterior part of the nose for a total dosage of 3,000 mg.-hr. She remained well until January, 1951, at which time she complained of pain in the right hip and iliac crest. Roentgenograms revealed a large osteolytic metastasis to the wing of the right ilium, proved by aspiration biopsy. She died of cancer in July, 1951, five and one-half years after her first visit.

CASE 5.—H. D., a 28-year-old, white, American-born woman, was first seen in the Stanford University Ear, Nose and Throat Clinic on Jan. 7, 1946, with a past history of having had nasal obstruction unsuccessfully treated in 1944 by a submucous resection. A tumor was found in the subcutaneous tissues of the right nasolabial fold encroaching upon the vestibule, which on biopsy was reported to be carcinoma, cylindroma type. She received 5,780 r to the tumor area, but the tumor recurred within one year and showed extension toward the inner canthus of the right eye and onto the right cheek. In October, 1947, a wide surgical excision of the right half of the nose and antrum was performed, but the tumor recurred in 18 months, just as plastic repair with a tube pedicle was being constructed. In June, 1949, she was treated with the chemosurgical technique by Dr. Frederick E. Mohs, at Madison, Wis., but the tumor recurred several months later and was then re-treated chemosurgically by Dr. R. A. Shepard, San Francisco, in November. She remained well until Aug. 23, 1950, at which time another recurrence was noted and treated by chemosurgery. Since that time and until May, 1951, she was never fully free of cancer. During May a chest roentgenogram revealed metastasis. The course has been steadily downhill, and she is dying of cancer at the present time (August, 1951), five years and eight months after her first clinic visit.

COMMENT

The similarity of symptoms in the first two patients presented is striking. An outstanding characteristic of the tumor, to infiltrate along nerve sheaths and nerve trunks, gave sensory loss which was an early presenting symptom in each. Its infiltrative character is illustrated by the widespread local invasion of bone and musculature, which in each patient caused orbital signs and symptoms as well as trismus. Pain was a late feature in one patient and negligible in the other.

The difficulty in obtaining an early diagnosis in the first case reveals the occult nature not only of antral cancer, but of an insidious, and in this case, slowly growing tumor. The difference in duration of clinical course emphasizes the variable life history. But there is no difference in the ultimate termination with widespread metastases which dramatically illustrate the malignant nature of this tumor.

The confusion in diagnosis in the second case, despite an adequate biopsy which pointed to the site and type of tumor, on hindsight appears incredible, but, at the time, the problem defied solution. The concomitant occurrence of a second tumor (ossifying fibroma, in itself a rare growth), which could have explained the physical findings, and the gross disappearance of the primary cancer, presumably by x-ray treatment, with regression of all physical signs, led to the assumption that the original diagnosis was an error.[10] Confusion was enhanced by the knowledge

10. Consideration might be given the theoretical possibility that the osteoma was the result of irradiation therapy which left as residual a fibrosis and ossification of the cancer and not, in fact, a new growth.

that these tumors are, in general, not responsive to irradiation, yet when the maxilla was removed, there was no gross evidence of cancer. The tumor dose given caused regression of the cancer, leaving the osteoma as the only evidence of disease. Autopsy verified the original diagnosis.

The patients whose tumors were nasal and nasopharyngeal in origin also showed differences in duration of clinical course, but with similar end-result. The difficulties of treatment in a surgically inaccessible area were enhanced by an invasive and infiltrative cancer which showed only moderate response to x-rays in the cases mentioned. The metastasis to bone in one patient again serves to illustrate the widespread nature of metastasis seen here with this type of tumor. The persistent and relentless nature of recurrences over a period of years, as illustrated by the final case, suggests a very guarded prognosis for all patients with this type of tumor, and it would suggest more radical measures as the initial form of treatment. Such measures tax the ingenuity of the surgeon because in this small series, at least, the majority (4 to 1) of patients were young women, all but one below 33 years of age.

SUMMARY

Cylindromas are malignant tumors occurring wherever salivary or mucous glands are found. They reveal a variable clinical course, but may metastasize widely to all viscera and to the skeleton, as demonstrated by the cases reported. The majority of patients in this small series were women in their twenties.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1949

CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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THE SUBJECT of otosclerosis and its surgical treatment continues to predominate in the literature on chronic progressive deafness. Labyrinthine hydrops is second from the standpoint of current interest. The material selected for review is arranged under the following heads: otosclerosis, pathologic studies and treatment; acoustic trauma and aero-otitis; hydrops of the labyrinth, and miscellaneous contributions on cause and treatment.

OTOSCLEROSIS

Pathologic Studies.—Lempert and Wolff [1] made detailed microscopic studies on 100 ossicles removed at fenestration operations. The changes of vascular content noted were the formation of clear globules, accumulation of fragmented red blood cells, sludge formation, and accumulation of polymorphonuclear cells. The vascular walls revealed constriction, tortuosity, periarteritis, and endarteritis. The bone changes noted were excessive blue mantling, hypervascularity, decalcification, osteofibrosis, osteoporosis, hyperostosis with malformations, ankylosis of joints, and ischemia with empty lacunas. It was believed that the vascular changes noted lead in more or less logical and chronological steps to the various types of bone disease described. The bony changes in the ossicles were thought to be otosclerosis, though not in the clear-cut circumscribed form usually seen in the otic capsule. The authors believe that

> the reason the lesions in the ossicles are not circumscribed by healthy bone, whereas in the otic capsule they are, is that the number of blood vessels normally feeding the ossicles is limited as compared with the much richer blood supply normally nourishing the otic capsule.

The initial pathologic changes in otosclerosis are believed by the authors to be vascular.

[Reviewers' Comment: The theory of Lempert and Wolff of the pathogenesis of otosclerosis is unique and interesting. Histologists may not all agree that the changes observed by the authors in these ossicles are otosclerosis. The question will be raised, "Are these changes definitely pathologic or are they within the limits of normal variability?" The answer will be found when these ossicles from otosclerotic ears have been compared with a similar series of ossicles from normal ears

1. Lempert, J., and Wolff, D.: Otosclerosis: Theory of Its Origin and Development, Arch. Otolaryng. **50**:115-155 (Aug.) 1949.

of the same age. Meanwhile, the conclusions reached by the authors that the vascular and bony changes seen in the ossicles represent an otosclerotic process must be regarded as tentative.]

Nylén [2] made an intensive study of the histologic changes in 74 cases of otosclerosis from the collection of temporal bones of F. R. Nager. The principal results of this histologic investigation were as follows:

Of the 74 cases investigated, with 121 temporal bones showing otosclerotic foci, 34 cases were of females and 40 were of males. The localization of the otosclerotic processes in 90% of the cases was the window regions. Approximately 50% of the cases with a localization at the oval window were accompanied with stapes ankylosis. The round window was involved in approximately 40% of the entire material, the cochlear capsule in 35%, the internal auditory canal region in 30%, and the semicircular canal capsule in 15%. Typical otosclerotic foci entirely outside the labyrinthine bony capsule were rare, being found in only two instances. In 55% the focus was limited to only one region. The process was confined to the stapedial region in 40% and to the round window in 8%. There was a higher proportional incidence of stapes ankylosis among the females.

The pathologic areas are usually symmetrically located in both temporal bones of a single case, except in cases of unilateral otosclerosis. A single focus occurred in approximately 65% of the total number of bones; in the others there were two or more foci, with more than three being rare. Classification of the foci according to their degree of activity showed active foci in 20% of cases, mixed, in 50%, and quiescent, in the remainder. Several foci present in the same bone or in the same person were usually of the same degree of activity. Mixed and quiescent areas were commoner in the group over 50 years of age. The individual focus measured 0.4 to more than 20 mm.

Diffuse otosclerosis occurred in approximately 10% of the cases. Stapes ankylosis was commoner in these, and the activity was extremely mixed.

The findings in the present investigation, as well as in other recent investigations, indicate that unilateral otosclerosis occurs in 15 to 30% of the cases in collections of histologic specimens.

Histologically, the extent of the foci in the oval window region was typical. The round window was often occluded by the process. The lumina of the cochlea, the vestibule, and the semicircular canals were often compressed, particularly in cases of diffuse otosclerosis, whereas foci in the internal auditory canal did not appear to compress the lumen to any extent.

[Reviewers' Comment: This paper is a valuable addition to our knowledge of the histologic picture of otosclerosis. It is interesting that the author noted frequent reduction of the lumen of the semicircular canals by the focus, whereas at operation large foci that include the bone of the horizontal semicircular canal do not appear to encroach upon the lumen as viewed through the operating microscope. The frequent occlusion of the round window is also of interest. In contrast with the clear-cut clinical picture of oval window occlusion, the clinical diagnosis of round window occlusion has not yet been clearly defined.]

2. Nylén, B.: Histopathological Investigations on the Localization, Number, Activity and Extent of Otosclerotic Foci, Upsala läkaref. förh. **54**:1-52 (March 15) 1949.

Fowler [3] studied the incidence and degrees of blueness of the scleras in a series of patients routinely examined. The incidence of blue scleras in those with otosclerosis was 69%, as compared with 10% in normal hearing controls, 16% in persons with nerve deafness, and 21% in those with otitis media deafness. Otosclerosis was diagnosed in 3% of those with white scleras; in 12% of those with questionably blue scleras; in 35% of those with two or more degrees of blueness; in 50% of those with three or more degrees of blueness, and in 100% of those with four degrees of blueness of the scleras.

[Reviewers' Comment: Fowler has presented a very interesting observation, confirmed by the reviewers. The exact significance of the definite association of blue scleras with otosclerosis is not known.]

Brunner [4] reports the microscopic temporal bone findings in a 41-year-old man who apparently was completely deaf. A reliable history concerning the onset of his deafness was not available. Both temporal bones revealed extensive otosclerotic changes and considerable osteoporosis. The stapes was fixed on each side. Some hyperostosis was present. There was an accumulation of connective tissue in both inner ears, with occasional small cysts, and frequently a network of spicules of calcium or newly formed bone was observed. Brunner speculates on the possibility of these inner ear changes being the result of the surrounding otosclerotic process but concludes that the labyrinthitis chronica ossificans in the present case must be considered purely coincidental.

Saltzman and Ersner [5] have previously observed that the use of a hearing aid usually "masks" tinnitus in cases of clinical otosclerosis. The patient reported on at this time experienced an increase in tinnitus when the aid was put on the poorer ear. Placing the aid on the better ear enabled the patient to "forget" the tinnitus in the other ear.

[Reviewers' Comment: The mechanism of tinnitus production in cases of otosclerosis is not known, except that tinnitus, as a rule, is severer in cases of advanced stapes ankylosis with cochlear nerve degeneration, suggesting that the same process that causes the nerve involvement causes the tinnitus. The mechanism of relief from tinnitus following fenestration is also unknown. Since severe tinnitus may be relieved without an appreciable gain in hearing, while other cases with a maintained hearing gain show a recurrence of tinnitus after a time, it does not seem likely that the masking effect noted by the authors always accounts for the improvement in tinnitus following fenestration.]

Treatment.—Lempert [6] reviews his observations on fenestration surgery based on 3,700 fenestrations, 450 revisions, and histologic studies of controlled animal research. He believes that the "endosteal bone cupola" technique which was described last year eliminates in a large measure the factors which contribute toward bony closure.

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3. Fowler, E. P.: The Incidence (and Degrees) of Blue Scleras in Otosclerosis and Other Disorders, Laryngoscope **59**:406-416 (April) 1949.

4. Brunner, H.: Otosclerosis Associated with Osteoporosis and Labyrinthitis Chronica Ossificans, Arch. Otolaryng. **49**:184-196 (Feb.) 1949.

5. Saltzman, M., and Ersner, M.: Tinnitus Aurium in Otosclerosis, Arch. Otolaryng. **50**:440-442 (Oct.) 1949.

6. Lempert, J.: Lempert Fenestra Nov-Ovalis for the Restoration of Practical Unaided Hearing in Clinical Otosclerosis: Its Present Status, J. Laryng. & Otol. **63**:57-69 (Feb.) 1949.

[Reviewers' Comment: The author's brilliant pioneer work in fenestration surgery is followed with intense interest by all otologists. As he has pointed out, the ultimate value of each modification of technique must await the test of time. The "cupola technique" gives promise of lessening the likelihood of bony closure due to bone dust particles entering or remaining in the fenestral opening.]

In order to determine whether release of perilymph pressure in fenestration is a factor in the hearing improvement obtained after surgical treatment, Lempert and associates [7] carried out several experimental studies. A minute cannula was inserted into the perilymph space of the lateral semicircular canal of a monkey, and electric potential readings from the round window area in response to sound stimulation were obtained with and without increased pressure in the perilymph. There was no decrease in potential with increase of perilymph pressure. The conclusion reached was that consequently a release of pressure after fenestration would not increase hearing. This experiment substantiates Lempert's observations at fenestration operations on human beings.

[Reviewers' Comment: The reviewers have done several revisions on fenestration cases in which the fenestra appeared to be wide open, even though the hearing had receded to the original preoperative level after an initial substantial gain. Reopening of the perilymph space by lifting the skin flap failed to produce any improvement in hearing. This is further evidence that increase in perilymph pressure is not the basic fault in deafness due to clinical otosclerosis.]

Meltzer and Lewis [8] present the results of 236 fenestration operations as performed by a "group of competent surgeons [who] gradually stabilized their proficiency in fenestration surgery" over a period of eight years. They classify their results into ideal, good, and borderline. Their best results were obtained in a recent group of cases in which the margins of the fenestra were burnished with lead. In these cases 77.7% have maintained a high degree of improvement after 13 months. They point out, however, that in these more recent cases the surgical technique had also undoubtedly improved as a result of experience and that this, no doubt, was also a factor in the improved results.

[Reviewers' Comment: This is a careful objective statistical study of results in a large series of fenestration operations which appear to indicate the value of lead burnishing. However, as the authors state, the improved results might be attributed to other improvements in technique. Moreover, results on the 13-month follow-up are probably still too early to warrant the drawing of final conclusions.]

Experimental studies on monkeys by Shambaugh [9] demonstrated six factors that influence osteogenic closure of the fenestra: the stimulating effect of bone dust particles on osteogenesis; the sluggish osteogenic response of the endochondral bone of the labyrinthine capsule as contrasted with the response of the periosteal bone; the inhibiting effect on osteogenesis of stratified squamous epithelium; the inhibiting effect on osteogenesis of intact endosteal membrane; the lessened osteo-

7. Lempert, J.; Weaver, E. G.; Lawrence, M., and Meltzer, P. E.: Perilymph: Its Relation to the Improvement of Hearing Which Follows Fenestration of the Vestibular Labyrinth in Clinical Otosclerosis, Arch. Otolaryng. **50**:377-387 (Oct.) 1949.

8. Meltzer, P. E., and Lewis, D. K.: Results of the Fenestration Operation, Tr. Am. Acad. Ophth. **53**:546-562 (May-June) 1949.

9. Shambaugh, G. E., Jr.: Fenestration Operation for Otosclerosis: Experimental Investigations and Clinical Observations in 2,100 Operations over a Period of 10 Years, Acta otolaryng., Supp. 79, pp. 1-101, 1949.

genic response from a smooth polished bone surface; the role of fibrosis in producing osteogenesis. Postoperative serous labyrinthitis was shown experimentally to be the result of blood and serum from the plastic skin flap entering the perilymph space at the fenestra and diffusing into the cochlea. To minimize osteogenic closure of the fenestra and postoperative serous labyrinthitis, a fenestration technique has been evolved utilizing an operating microscope with a continuous flow of isotonic sodium chloride solution to render the fenestra immaculately clean of bone dust particles; the fenestra is constructed on top of a dome in order to surround it with a wide zone of relatively inactive endochondral bone and to insure a closely fitting skin flap; the margins of the fenestra are left sharp with intact endosteal membrane that will adhere to the periosteum of the skin flap; the bone around the fenestra is burnished with a smooth hard gold burr; great care is taken to maintain perfect asepsis and hemostasis. The results obtained by the author and his associates in a series of cases with operation by this technique are summarized in detail, with respect to both the decibels of hearing gained and the hearing level attained.

Shambaugh and Juers [10] summarize the fenestration technique which has been evolved at the Northwestern University Medical School, Chicago. Freeing most of the meatal skin flap before entering the mastoid cortex is a recent change in technique. Particular stress is placed on the desirability of contouring the fenestral margin in a manner that will provide intimate contact between the endosteum and the overlying flap. The most recent statistical study [9] indicates that 80% of those with ideal suitability will maintain a practical level of hearing or better at the end of the first postoperative year. The level of hearing of 5% of these will drop somewhat during the second year, but an improvement maintained two years is almost certain to be permanent as far as subsequent closure of the fenestra is concerned.

Woodman [11] reports his general observations on a series of 75 fenestrations. It is his impression that there is a definite early improvement of hearing in the untreated ear. This effect seems to be a central tonic response to the stimulation caused by the fenestration operation and is compensatory and transient. The few cases of bone-conduction gain of lasting nature are explained as being coincidental.

[Reviewers' Comment: It has been the consistent experience of the reviewers in approximately 3,000 fenestration operations that the hearing in the untreated ear does *not* show a significant gain or loss immediately after operation, except in the comparatively rare case of psychogenic overlay, in which a marked improvement may occur in the untreated, as well as in the treated, ear.]

Farrior [12] has recently used general anesthesia for the fenestration operation and believes that it possesses definite advantages over local. The general technique followed is that described by Shambaugh. A primary skin graft is used in the cavity. Of 100 fenestrations with follow-up ranging from 12 to 33 months post-

10. Juers, A. L., and Shambaugh, G. E., Jr.: The Evolution of the Northwestern University Fenestration Technic, Quart. Bull. Northwestern Univ. M. School **23**:56-60 (Spring) 1949.

11. Woodman, DeG.: The Fenestration Operation for Otosclerosis: A Survey of Experience with 75 Cases Together with a Study of the Effect of the Contralateral Ear, Laryngoscope **59**:1299-1319 (Dec.) 1949.

12. Farrior, J. B.: The Fenestration Operation: Indications, Technique and Results, Laryngoscope **59**:515-539 (May) 1949.

operatively, he estimates that in 82% hearing satisfactory to the patient has been maintained. He stresses the value of auditory training after surgical treatment.

Of 100 patients in whom Fasse [13] used the stopple technique, only 42 maintained improved hearing. In 379 patients operated on between 1946 and 1948, he used the technique of endochondralization, irrigation, and microscopic visualization. These cases have been followed one to three years. Sixty-eight per cent of the class "A" patients (good cochlear function) have reached and maintained a level of 30 db. or better. In order to prevent excessive compression of the membranous labyrinth, he now leaves a little more of the medial (superior) margin of the fenestral rim to serve as a "hood."

Hall [14] reviews his present fenestration technique. He still prefers the postauricular approach. He believes that producing contact between the membranous labyrinth and skin flap over a wide area is the best means of preventing bony closure.

[Reviewers' Comment: The success of the fenestration operation is dependent upon the perfection of the technique of making the fenestra. The author has long used continuous irrigation and an operating microscope for this essential part of the operation. His results are evidence that the approach one uses, that is, endaural or postaural, is immaterial.]

Popper [15] describes the current technique for his transmeatal transtympanic approach to temporal bone operations involving only the middle ear and attic area. The incision may be antetragal or a modified endaural one. The details of the technique can be understood only by studying the drawings in the original article. With reference to radical mastoidectomy, he states that "after the incus has been removed during the radical mastoidectomy the hearing that was present on the affected side is gone."

[Reviewers' Comment: The author's last statement is not always true. One of the reviewers (A. L. J.) has observed a patient with an attic cholesteatoma for which a modified radical mastoidectomy was done and the incus found to be absent. Nevertheless, practical hearing remained after surgical intervention. Another otologist [16] has observed a similar case. There are undoubtedly others unreported. Preservation of an intact middle ear cavity is, of course, essential to retaining good hearing in these cases.]

Carruthers, in discussing the above paper, stated that he had used both the Lempert endaural and the Popper transmeatal approach, and the hearing results obtained by the latter appeared to be equal to those obtained by the former. However, he stated that in those cases in which Popper's approach had been used he had been

> . . . troubled much with excessive granulations in the depth of the canal, and later stenosis has been too frequent after this method has been used. I believe that the tendency toward these unsatisfactory sequelae points to the necessity to make the bony atticomastoidectomy much more extensive and indicates also that the tympanomeatal flap should be much larger.

13. Passe, E. G.: A Second Interim Report on the Result of Over 500 Cases of the Fenestration Operation, J. Laryng. & Otol. **63**:495-500 (Sept.) 1949.

14. Hall, I. S.: On the Technique of the Fenestration Operation, J. Laryng. & Otol. **63**:382-389 (July) 1949.

15. Popper, O.: Transtympanic Approach and Fenestration: II. Current Technic, Arch. Otolaryng. **49**:350-379 (April) 1949.

16. Boies, L. R.: Personal communication to the reviewers.

[Reviewers' Comment: If the last two modifications of Popper's technique are made, then one has essentially returned to the Lempert endaural approach, in which the mastoid cavity can be kept very small.]

Juers takes a rather critical point of view in discussing Popper's technique. He points out that the regular Lempert endaural technique can be modified by cutting the flap and freeing it down to the tympanic ring before the bony cortex is entered with a burr. Hence the meatus can be utilized in the endaural approach to the lateral semicircular canal. Minimal removal of mastoid structure is necessary. Failure to remove the entire lateral antral wall in Popper's technique leaves a recess in the antrum area which will undoubtedly in some instances eventually result in a cholesteatomatous formation. Furthermore, maximum hearing improvement cannot be obtained if the small flap advocated by Popper fails to seal completely the middle ear cavity. Dissections of the labyrinth reveal that it is anatomically impossible to place the fenestra any further anteriorly than can be done via the usual endaural approach.

Lund [17] reports results of operations on 90 patients by Popper's approach with a few modifications intended chiefly to avoid atresia of the external meatus. He uses Shambaugh's endochondralization technique in creating the fenestra. The hearing tests carried out by Ewertsen reveal that of those followed more than six months, 63% showed improved hearing (more than 5 db. on pure-tone audiometry). Among the better-suited patients, 75% obtained definite improvement. In 4 of 30 cases the fistula sign changed from positive to negative with a drop in hearing. Tinnitus was relieved in about half of the patients. Rasmussen tested the vestibular function before and after operation in 79 patients. Twenty-seven per cent of the patients had been suffering from vertigo before the operation. This symptom was present after operation in 64 of the 79 patients. The fistula sign was originally present in 57 patients but later disappeared in 11. Biochemical studies of serum values for calcium, inorganic phosphate, phosphatase, total cholesterol, albumin, and globulin were carried out by Riskaer. No definitely abnormal values were found.

Tucci [18] compared the results of heavy preoperative sedation in one group of fenestration operations with those of a combination of lighter sedative premedication and either nitrous-oxide-oxygen-ether administration or a barbiturate given intravenously in another group. Regional anesthesia was used in both groups. From every standpoint the combination used in the second group seemed to be most satisfactory because of less depression and better patient control during surgical procedures. There was no increase in bleeding.

The administration of dimenhydrinate (dramamine®) to 28 patients during the first two days after fenestration was observed by Campbell [19] to diminish the usual early vestibular symptoms.

Jongkees and Hulk [20] studied the vestibular function before and after fenestration in 25 patients. All patients showed a spontaneous nystagmus toward the untreated

17. Lund, R.; Ewertsen, H.; Rasmussen, H.; Riskaer, N., and Selsø, S.: Acoustic, Vestibular and Other Problems Concerning Otosclerosis and Its Surgical Treatment According to Popper's Method, Arch. Otolaryng. **49**:380-392 (March) 1949.

18. Tucci, J. H.: Anesthesia in Fenestration Surgery, Ann. Otol. Rhin. & Laryng. **58**:61-69 (March) 1949.

19. Campbell, E. H.: The Effectiveness of Dramamine in Relieving the Vestibular Reactions Following the Labyrinthine Fenestration Operation, Laryngoscope **59**:1260-1272 (Nov.) 1949.

20. Jongkees, L. B. W., and Hulk, J.: Changes in Vestibular Reactions After the Fenestration Operation, J. Laryng. & Otol. **63**:225-232 (March) 1949.

side for several days after surgical intervention. In 30% the direction changed after a few days to the side operated on. Cupulograms returned to normal preoperative levels after about two months. The strength of the galvanic current needed to produce a nystagmus was diminished for the treated ear and increased for the untreated ear. There was no simple connection between the gravity of the objective vestibular symptoms and the subjective vertigo.

Rosen [21] studied the position of Shrapnell's membrane with relation to the fenestra in 50 fresh cadaver operations. In no instance did Shrapnell's membrane cover the fenestra.

[Reviewers' Comment: In all probability the fenestra is covered with periosteum in the great majority of cases. Periosteum devoid of bone particles acts as a limiting membrane and helps to inhibit rather than promote osteogenic closure. It is essential that the periosteal surface of the skin flap be examined minutely under magnification to discover and remove all scales and spicules of bone that would act as foci for new bone formation.]

ACOUSTIC TRAUMA AND AERO-OTITIS

Rice [22] reports his observations on a series of 87 cases of blast perforation of the eardrum membrane. Twenty-one membranes healed without assistance. Patches of processed sheep's cecum were placed over 59 perforations. In 43 of these the drum membrane healed under the patch, and in 16 healing did not take place during the period the ears could be observed. Seven membranes would not retain a patch and also failed to heal. The most striking observation was the rapidity with which a damaged drum membrane healed when an acute febrile infection of the middle ear supervened.

Blast injuries to the ear following the Texas City disaster are discussed by McReynolds, Guilford, and Chase.[23] The observations were made on 143 patients who were hospitalized for more serious surgical injuries. There were 77 tympanic membrane perforations, 11 of which had not healed after 30 days. All patients were given penicillin. The ear canals were cleaned under sterile technique without irrigation. A small amount of sterile sulfadiazine powder was insufflated. True suppurative otitis media developed in only one instance. Those closest to the blast sustained the greatest trauma. Intervening structures offered some degree of protection from the positive phase of the blast wave.

Trowbridge [24] describes an aspiration technique for the immediate relief of the symptoms of acute aero-otitis media. This technique may also be used for the removal of other types of nonpurulent tympanic secretions.

Davis and associates [25] find no evidence to indicate that air-borne ultrasonic vibrations constitute a practical hazard to hearing or produce any specific effects

21. Rosen, S.: Is It Possible to Cover the Fenestra Nov-Ovalis with Shrapnell's Membrane? Arch. Otolaryng. **49**:528-535 (May) 1949.

22. Rice, A. H.: Blast Perforation of the Ear Drum, Arch. Otolaryng. **49**:316-331 (March) 1949.

23. McReynolds, G. S.; Guilford, F. R., and Chase, G. R.: Blast Injuries to the Ear: The Texas City Disaster, Arch. Otolaryng. **50**:1-8 (July) 1949.

24. Trowbridge, B.: A New Treatment of Acute Aero-Otitis Media, Arch. Otolaryng. **50**:255-263 (Sept.) 1949.

25. Davis, H.; Parrack, H., and Eldredge, D. H.: Hazards of Intense Sound and Ultrasound, Ann. Otol. Rhin. & Laryng. **58**:732-738 (Sept.) 1949.

on the nervous system or sense organs. A frequency of 6,500 cps at 159 db. caused nearly intolerable pain to one subject and ruptured his tympanic membrane during a five-minute exposure. Some high-tone hearing loss above 10,000 cps persisted after 14 weeks.

Senturia [26] concludes that a fairly large percentage of the men in pilot training show temporary hearing losses following a period of exposure to aircraft noise. These temporary losses tend to involve the frequencies 1,024 to 5,792. In the more vulnerable 2,896 to 5,792 regions, the recovery of hearing losses is delayed, resulting in some increased "V" notching. However, on the whole, the hearing losses incurred during 210 hours of flight training are temporary losses which are partially or completely recovered after 24 hours of rest. So far as pilot training is concerned, the prediction that large numbers of flying personnel would be permanently deafened in World War II has not been substantiated.

Most previous investigations into the question of occupational hearing loss in railway employees have led to the conclusion that working on an engine is injurious to the hearing. Since most of these studies were made before the audiometer was available, Lumio [27] studied the hearing, including the audiograms, in 203 locomotive engineers and firemen. Comparing the average audiograms by decades for these engine employees with the average audiograms for Finns of the same age group and free from ear disease, the author finds a definite decline in the hearing of the engine employees as compared with the normal persons. Comparison of the average audiograms for both groups according to the years of work showed a similar loss, greater than would be expected without exposure to noise. However, the average decline in hearing was not great when considered according to either age or years of work.

Dickson and Watson [28] made observations on the effect of turbo-jet engine noise on the auditory apparatus and central nervous system. The source of noise is the engine turbine and compressor and the disturbance created as the craft passes through air at a high velocity. In a third of the personnel a short period of discomfort in the ears, a minor degree of deafness, and tinnitus were noted after the "running up" of the engine. Audiometrically no deterioration of the hearing was observed during the year of the study. Electroencephalograms revealed no significant effect upon the cerebral electrical activity.

HYDROPS OF THE LABYRINTH

Lathrop [29] reviews observations on 91 patients who had section of the eighth nerve for relief of vertigo due to hydrops. Twenty-five had total section. Vertigo was completely relieved in 15 and remained unchanged in 4. One had a slight unsteadiness. Five could not be traced. In 66 patients the resection was confined to the vestibular component of the eighth nerve. Forty of these 66 patients were

26. Senturia, B. H.: Hearing Changes During Pilot Training, Mil. Surgeon **105**:205-208 (Sept.) 1949.

27. Lumio, J. S.: Studies on Hearing Loss of Railway Engine Employees in Finland, Acta oto-laryng. **37**:539-550, 1949.

28. Dickson, E. D. D., and Watson, N. P.: A Clinical Survey into the Effects of Turbo-Jet Engine Noise on Service Personnel: Some Preliminary Remarks, J. Laryng. & Otol. **63**:276-285 (May) 1949.

29. Lathrop, F. D.: Surgery of Ménière's Syndrome: Evaluation of Neurosurgical and Otologic Procedures, Ann. Otol. Rhin. & Laryng. **59**:809-824 (Sept.) 1949.

completely relieved of vertigo; 5 noted no improvement, and 6 were unsteady with no real vertigo. Thirteen could not be traced. Two patients died postoperatively, one of ependymitis and the other of suicide on the sixth day. Postoperative audiograms were made of 10 patients, and the hearing in the ear operated on was improved in 2, unchanged in 4, and worse in 4, as compared with preoperative tests. Electrocoagulation of the labyrinth was done in 14 patients. The vertigo was completely relieved in all but one, and this patient stated it no longer resembled the vertiginous attacks she experienced before surgical treatment. There were two instances of facial paralysis after operation—one immediate and one delayed. Recovery was almost complete four months after operation. The hearing was totally lost in all cases. Tinnitus was completely relieved in three, improved markedly in five, remained unchanged in four, and was worse in one.

[Reviewers' Comment: This study indicates that labyrinthotomy according to Day's technique is preferable to intracranial section of the eighth nerve when the hearing is good in the opposite ear and poor in the affected ear. In those rare cases in which it is important to preserve hearing in the affected ear, intracranial section of the vestibular nerve is indicated.]

Lindsay [30] reviews the surgical procedures which have been developed for the relief of intractable Ménière's disease. With the hope of relieving the vertigo but preserving the hearing, he did a simple fenestration on three cases. In two an attempt was made to cut the ampullary nerve without injuring the membranous canal. The fenestra was left uncovered and the mastoid incision closed completely. A Tullio phenomenon was present in all after surgical treatment, and a secondary destructive type of operation was necessary in two cases to relieve the persistent vertigo. At the secondary operation the original fenestra was found fully patent. A fourth patient had had a previous radical mastoidectomy and a fenestration, and cutting of the ampullary nerve was done. The hearing was worse after surgical intervention, but the vertigo was almost completely relieved. Lindsay concludes that this type of operation is not satisfactory.

Day and Lindsay [31] made a microscopic study of the temporal bones from a patient on whom Day several years previously had done an electrocoagulation type of operation for Ménière's syndrome. The hearing was preserved and vertigo relieved. The untreated ear showed a normal endolymphatic system and normal cochlear duct, stria vascularis, and Corti's organ. The treated ear presented the usual picture of endolymphatic hydrops. The coagulation procedure had destroyed the ampullae and all the proximal limbs of the horizontal and superior vertical canals and the utricle. New bone and fibrous tissue replaced these structures. Corti's organ and the tectorial membrane were fused together in the basal and most of the middle coil, with an apparent obliteration of hair cells. In the upper coil the organ of Corti could be more clearly distinguished, and in some slides the presence of hair cells, as well as their supporting cells, seemed fairly definite. The conclusion seemed to indicate that the final hearing level of about 15 db. in this ear may have occurred without the aid of sensory cells in the basal and middle coils.

[Reviewers' Comment: The undoubtedly near-normal hearing of the affected ear, in the presence of marked degeneration of Corti's organ in the basal and middle

30. Lindsay, J.: Labyrinthine Surgery for Ménière's Disease, Laryngoscope **59**:22-34 (Jan.) 1949.

31. Day, K. M., and Lindsay, J.: Hydrops of the Labyrinth: Case Report, Coagulation Operation, Clinical Course and Histopathology, Laryngoscope **59**:213-227 (March) 1949.

coil, is difficult to reconcile with what is known of the physiology of hearing and suggests that much remains to be learned of the function of the cochlea.]

Cawthorne [32] observed that in all labyrinthotomies done for Ménière's disease there was complete loss of cochleovestibular function. Cochleovestibular function was not affected in three of five incisions of the saccus endolymphaticus (Portmann's operation). The cochlear function was made worse in two fenestrations for otosclerosis in which the membranous labyrinth was torn at the posterior edge of the fenestra.

[Reviewers' Comment: The reviewers have observed through the operating microscope cases of undoubted perforation of the endolymphatic labyrinth during fenestration that did not result in loss of cochlear function. Perhaps the hemorrhage that occurs with removal of the endolymphatic labyrinth from the semicircular canal in Cawthorne's procedure is responsible for the loss of cochlear function.]

Hilger [33] correlates the many factors influencing the autonomic balance of the vascular structures of the inner ear. He accounts for inner ear disturbances which are not on an inflammatory basis as being largely the result of vasospasm of a small or large arterial segment. Symptoms will be dependent on the function of the structures involved in the vasospastic phenomenon and the severity of the resulting increase in capillary permeability. He uses the term vasomotor labyrinthitis when all components of the inner ear are involved, and the term vasomotor cochlearitis or vasomotor vestibulitis when only acoustic or only static components have yet been involved. The stability of the autonomic balance is largely a matter of heredity. Further imbalance of an unstable autonomic system may be the result of a single factor or the summation of several. These factors are sensitivity to food or inhalant substances, physical environment, such as temperature fluctuations and humidity, and, perhaps most important of all, emotional disturbances. He believes that endolymphatic hydrops is the end-result of vasospastic episodes. The efficacy of vasodilators, such as nicotinic acid and histamine, is thus explained.

Brown [34] reports additional case histories of the tendency of Ménière's syndrome to occur in more than one member of a family. Migraine is frequently associated with endolymphatic hydrops or is prominent in the family history. This is considered to be further evidence of a tendency toward poorly regulated blood vessels in these persons.

[Reviewers' Comment: The conclusion reached by Brown would substantiate Hilger's concept that the basic disturbance in Ménière's syndrome is neurovascular and that the state of balance of the autonomic nervous system is to some extent a matter of heredity.]

Asherson [35] feels that Ménière's syndrome rarely develops before the age of 50, at which time the presbycusic ear changes occur. He believes that the causation of both of these conditions is based on vascular changes and discusses the labyrinthine arterial distribution as described in detail by Nabeya.

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32. Cawthorne, T.: The Effect on Hearing in Man of Removal of the Membranous Lateral Semicircular Canal, Acta oto-laryng., Supp. 78, pp. 145-149, 1949.

33. Hilger, J.: Autonomic Dysfunction in the Inner Ear, Laryngoscope **59**:1-11 (Jan.) 1949.

34. Brown, M.: The Factor of Heredity in Labyrinthine Deafness and Paroxysmal Vertigo (Ménière's Syndrome), Ann. Otol. Rhin. & Laryng. **58**:665-670 (June) 1949.

35. Asherson, N.: Aural Vertigo (Ménière's Syndrome): An Audiographic Survey, J. Laryng. & Otol. **63**:520-530 (Sept.) 1949.

[Reviewers' Comment: The hearing loss of labyrinthine hydrops is typically a flat audiometric curve, with marked fluctuations. On the contrary, presbycusis is typically a high-tone loss without significant fluctuations. In this, as well as in other respects, the two conditions are so dissimilar that it seems unlikely that they are due to a similar cause.]

On the basis of recent observations, Atkinson [36] states that patients with Ménière's syndrome experience two distinct types of vertigo, rotational and positional, which have been found to be associated with different and specific vitamin deficiencies. Patients who experience rotational vertigo alone show signs of nicotinic acid deficiency in the tongue, give a small response to histamine injected intradermally, and can be relieved of their attacks with administration of nicotinic acid. Nicotinamide is ineffective in this group unless given in very large doses. Those patients who experience positional vertigo alone show signs of riboflavin deficiency in the tongue, the eyes, and the skin, give a large response to histamine injected intradermally, and can be relieved of their attacks by administration of riboflavin or histamine. The majority of patients experience both kinds of vertigo, show signs of a deficiency of both fractions, give an intermediate response to intradermally injected histamine, and can be relieved of their attacks only if both vitamin B fractions are given. General deficiency symptoms, such as fatigue, irritability, and palpitations, are frequently relieved by treatment of the associated thiamin deficiency. Therapy by the intravenous route is most effective during the acute episodes. Too large initial dosage sometimes aggravates symptoms.

Atkinson [37] discusses various aspects of the cochlear disturbance in 87 consecutive cases of Ménière's syndrome. There were 123 ears with impaired hearing in this group of patients. The deafness was of the purely perceptive type in 50%, mixed in 43%, and of the purely conductive type in 4%. There were four "dead" ears in this group. There was bilateral involvement in 43%. It seemed that the conductive type of deafness and positional vertigo showed greater preponderance in the vasodilator or intermediate histamine groups in which riboflavin deficiency predominates. Rotational vertigo and perceptive vertigo were commoner in the nicotinic-acid deficient group. With appropriate intensive vitamin therapy hearing was restored to normal in 3 of 101 affected ears. Improvement of 10 db. or more was achieved in 21 ears, the results being better in the nicotinic-acid-deficient group. The four dead ears were in the riboflavin-deficient group. No ears with pure conductive deafness showed any improvement. Fifteen per cent were completely relieved of tinnitus, and 51% improved.

Selfridge [38] reviews the more recent literature with particular reference to the etiology of Ménière's symptom complex and states that nutritional factors, such as electrolytes and vitamins, are particularly significant. Additional consideration must be given to endocrine substances, psychosomatic problems, and factors of physical environment. As these various etiological aspects are better understood, the surgical approach to therapy has become less popular.

[Reviewers' Comment: Because of the tendency toward spontaneous exacerbations and remissions, as emphasized by Lindsay, it is difficult to evaluate the results

36. Atkinson, M.: Ménière's Syndrome: Observations on Vitamin Deficiency as the Causative Factor; I. Vestibular Disturbance, Arch. Otolaryng. **49**:151-175 (Feb.) 1949.

37. Atkinson, M.: Ménière's Syndrome: Observations on Vitamin Deficiency as the Causative Factor; II. The Cochlear Disturbance, Arch. Otolaryng. **50**:564-588 (Nov.) 1949.

38. Selfridge, G.: Ménière's Symptom Complex, Arch. Otolaryng. **49**:1-15 (Jan.) 1949.

of therapy in Ménière's disease, and one must be guarded in attributing improvement to vitamins or other treatment administered during an exacerbation of symptoms.]

MISCELLANEOUS CONTRIBUTIONS ON CAUSE AND TREATMENT

Siirala [39] reports a case of unilateral congenital atresia in which he did a fenestration because the surgical procedure of Pattee did not produce an improvement in hearing. The cavity was lined with a special postauricular flap and Thiersch grafts. The hearing improved to the 35 db. level. The author states that this, to his knowledge, is the first case to be reported in which a fenestration was carried out for congenital atresia.

[Reviewers' Comment: Ombredanne has reported doing fenestrations on congenital atresias. This has been reviewed in previous years.]

Leisti [40] compared the auditory acuity of 451 persons, aged 16 to 92, not engaged in noisy work, with that of 40 young normal persons as controls. Beginning with the third decade, there was progressive loss with each decade. The loss for high frequencies as determined by pure-tone audiometry was approximately twice as much as for the low frequencies. The loss in the eighth decade varied from 22 db. at the 128 frequency to 66 db. for the 8,192 frequency.

Simpson [41] studied 100 cases of perceptive deafness at the Hull Royal Infirmary, Hull, England, for analysis of the primary factor behind the usually given causes. Congenital cases were not included. The cases are grouped under the following primary causes:

1. Neuritic—primary involvement of the nerve elements of the perceptive mechanism, such as that caused by influenza, measles, and mumps, 10 cases

2. Traumatic—skull fracture, blast injuries, occupational exposure, 30 cases

3. Vascular—Ménière's disease, arteriosclerosis, anemia, cerebral thrombosis, chronic otitis media, syphilis, 60 cases

He proposes the concept that the 4,096 dip in trauma cases may actually represent a localized reflex vascular reaction in response to the stimulation by intense sound. If this is true, the traumatic group would also be included in the vascular.

Saltzman and Eversden [42] made an etiological classification of 1,000 deaf patients presenting themselves for rehabilitation by means of hearing aids. Sixty-six and two-tenths per cent had a perceptive type of deafness. Fourteen per cent were classified as having mixed deafness. Otosclerosis was the etiological factor in 16.9%. A psychogenic overlay on an organic type of deafness was encountered rather frequently.

Hopkins and Kinzer [43] studied a group of 92 infants with deafness due to rubella in the mother and related the degree of developmental arrest with the month

39. Siirala, U.: Plastic Correction of Congenital Atresia of the External Auditory Meatus Combined with Fenestration to Improve Hearing, Acta oto-laryng. **37**:307-314, 1949.

40. Leisti, T. J.: Audiometric Studies of Presbyacusis, Acta oto-laryng. **37**:555-562, 1949.

41. Simpson, R. R.: The Causes of Perceptive Deafness, Proc. Roy. Soc. Med. **42**:536-540 (July) 1949.

42. Saltzman, M., and Eversden, M.: A Statistical Study of Incapacitating Hearing Impairment in 1,000 Clinic Patients, Ann. Otol. Rhin. & Laryng. **58**:345-350 (June) 1949.

43. Hopkins, L. A., and Kinzer, L. G.: Rubella-Deafened Infants, Am. J. Dis. Child. **78**: 182-200 (Aug.) 1949.

of pregnancy during which the rubella occurred. In the majority the infection took place in the second month of pregnancy. The third month was next, and the fewest were in the first-month group.

Hiller [44] studied 42 congenitally deaf children in the Tasmania Institute for the Blind and Deaf. In 32 of these children the history indicated that the deafness was due to rubella in the mother early in pregnancy. The average speech frequency loss varied from 81 to 100 db. In general, the rubella-deafened children had a somewhat flat curve and the other children had a more precipitate loss above the 256 frequency.

A case of psychogenic deafness in a 9-year-old boy is reported by the Children's Psychiatric Service of the Massachusetts General Hospital, Boston.[45] This boy was sent to the psychiatric service for investigation of a behavior problem before being fitted with a hearing aid. In reviewing his clinical record, it was noted that his results on tests had been somewhat inconsistent at times. After gaining the boy's confidence, a retest revealed a normal audiogram, indicating that his temporary hearing loss was on a psychogenic basis.

Cogan [46] reports four additional cases of nonsyphilitic interstitial keratitis with loss of function of both the vestibular and the cochlear portions of the inner ear. In a few cases the loss of labyrinthine function was incomplete. No causation could be established. One patient had an eosinophilia, but no positive reactions to intradermal tests with the common allergens were obtained.

Dix and associates [47] report further on the differential diagnosis of Ménière's disease and eighth nerve neurofibroma. Loudness recruitment was always observed in Ménière's disease, but not at all in neurofibroma. With word-intelligibility tests, in Ménière's disease it was observed that as loudness increased intelligibility decreased, whereas in neurofibroma intelligibility continued to improve with amplification up to high levels. In other words, the results of these tests in cases of neurofibroma corresponded to the results usually obtained only in cases of conduction deafness.

Ireland [48] reviews the preoperative findings in 92 cases of proved acoustic neuromas operated on from 1924 to date. Perceptive deafness was usually the earliest symptom, with tinnitus associated in about half. There were four cases with partial nerve deafness and complete loss of caloric response and two cases with no nerve deafness, but absent caloric response. Of the cases with a record of satisfactory response on caloric test, 26 showed a complete loss and 2 a partial loss. No cases tested had a normal caloric response. Symptoms of true peripheral type of vestibular disturbances are rare in these cases because of the gradual development of compensation for the vestibular hypofunction. The sensory division of the fifth nerve is the commonest other cranial nerve to be affected early, as shown

44. Hiller, B.: Rubella Congenital Inner Ear Deafness in Tasmania, M. J. Australia **2**:277-283 (Aug.) 1949.

45. Psychogenic Deafness in a Disturbed Boy: Clinic on Psychosomatic Problems, Am. J. Med. **7**:221-227 (Aug.) 1949.

46. Cogan, D. G.: Nonsyphilitic Interstitial Keratitis with Vestibuloauditory Symptoms, Arch. Ophth. **42**:42-49 (July) 1949.

47. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: "Nerve" Deafness: Its Clinical Criteria, Old and New, Proc. Roy. Soc. Med. **42**:527-536 (July) 1949.

48. Ireland, P. E.: Otological Findings in Acoustic Nerve Tumors, Ann. Otol. Rhin. & Laryng. **58**:716-731 (Sept.) 1949.

by a diminished corneal reflex. Cerebellar and visual symptoms usually follow. Facial paralysis occurred in a moderate number of cases but was usually mild.

[Reviewers' Comment: Acoustic neuroma should be suspected in any case of unilateral perceptive deafness of gradual onset, associated with partial or complete loss of caloric response.]

Jones [49] describes his management of remedial deafness in children, principally that produced by tubal occlusion. A thorough tonsillectomy and adenoidectomy should be done first when indicated. Radium therapy has been of limited value. Treatment of sinusitis and allergy is essential. Either or both of these factors may contribute toward recurrence of lymphoid tissue in the nasopharynx. Adult patients with impaired hearing frequently give a history of recurrent or continuous colds during childhood. Undoubtedly allergy is an important factor in these cases.

[Reviewers' Comment: The reviewers concur with the author's emphasis on an underlying allergy in cases of tubal occlusion in children.]

Lymphoid tissue obstructing the Eustachian tube should, in Meltzer's [50] opinion, be removed surgically under direct visualization of the nasopharynx. In his experience the nasopharynx can be viewed directly only with the patient in the upright position. By the use of intratracheal anesthesia, the trachea can be kept free of blood. Special basket punches are used to remove the follicles of lymphoid tissue in the Eustachian tube area. He believes that radiation should not replace surgical measures, but that radiation therapy should be limited to patients with persistent recurrence of otitis media who have had proper surgical treatment and in whom the remaining lymphoid tissue cannot be removed by further surgical procedures. Mastoid surgery with respect to conservation of hearing is discussed.

[Reviewers' Comment: The reviewers agree with the author that radiation *is not* a substitute for adenoidectomy but should be reserved as an adjunct following a competent complete adenoidectomy for those patients that continue to have tubal occlusion and recurring otitis media.]

Rosenberger [51] feels that roentgen irradiation is preferable to use of the radium applicator when radiation therapy is selected in a case of tubal obstruction due to lymphoid tissue because the radiation from the applicator fails to reach the deeper portions of the tube and the surrounding structures which may contain lymphoid centers. Of 21 patients treated, only 7 showed significant improvement of hearing. One disappointing feature was the recurrence of aural symptoms with subsequent colds.

Fowler and Feind [52] present a series of experiments on cats to whom streptomycin was given to determine whether the toxicity of streptomycin could be lessened (1) by desensitization through the giving of small doses of streptomycin with fairly rapid increases in the dosage to both toxic and therapeutic levels; (2) by administration of streptomycin plus an antihistamine, and (3) by desensitization plus

49. Jones, M. F.: The Management of Ear Problems in Children, New York J. Med. **49:** 2825-2828 (Dec.) 1949.

50. Meltzer, P. E.: Surgical Procedures Directed Toward the Prevention of Hearing Impairment in Children, Laryngoscope **59:**1225-1237 (Nov.) 1949.

51. Rosenberger, H. C.: Radiation Therapy for Conductive Deafness, Arch. Otolaryng. **49:**504-510 (May) 1949.

52. Fowler, E. P., Jr., and Feind, C. R.: Toxicity of Streptomycin for the Auditory and Vestibular Mechanisms, Acta oto-laryng., Supp. 78, pp. 193-197, 1949.

administration of an antihistamine. A control group of 14 cats was given streptomycin in varying levels and doses. The animals on the desensitization regimen tolerated almost twice as much drug as the controls. The antihistamine group was about midway between the control and the desensitized group. One disadvantage of the desensitization regimen is that it cannot be used in acute lesions and may also enable the infecting organism to acquire a tolerance to the drug.

Winston and co-workers [53] studied the toxic effects of streptomycin on the central nervous system of cats. The trypan-blue vital-staining method revealed pathologic changes in the cerebellum and medulla in 50% of the animals that were given streptomycin in three divided daily doses and in none of those receiving the drug once a day. With the use of routine histologic stains, evidence of pathologic change was found in 59.2%. Surgical destruction of the vestibular nuclei on one side failed to induce nystagmus such as would result from a similar procedure carried out on a normal animal. This was interpreted as further evidence of a central toxic effect, although it would not preclude the possibility of concomitant damage to the peripheral vestibular end-organs.

Berg [54] conducted some controlled experiments on 25 cats to determine histologically the site of the toxic effect of large doses of streptomycin on the eighth cranial nerve. Postrotary nystagmus was absent in all animals on completion of the period of medication, indicating loss of vestibular function. The histologic study revealed that the streptomycin affects primarily the sensory epithelium of the labyrinth. Degenerative changes in the organ of Corti were observed in only one specimen. Berg believes that the lesser changes noted in the vestibular ganglion and in the nucleus of Deiters are secondary to the primary lesion of the sensory epithelium.

Wallner [55] studied the toxic effects of streptomycin therapy on 93 patients with tuberculosis. Fifty-three patients in Group I received 2 gm. per day for four months. Of these, 42 noticed subjective symptoms of dizziness, staggering gait, or visual disturbances, and 25 had objective evidence of depressed or absent vestibular function. Group II consisted of 40 patients who received 1 gm. per day. Of these, 18 complained of subjective symptoms and 13 had depressed or absent caloric responses. Partial compensation of the vestibular disturbances occurred during the period of observation. The only patient to have any loss of hearing was one with a temporary anuria who undoubtedly sustained a high concentration of the drug. He suffered almost total loss of hearing.

Peik, High, and Campbell [56] observed the toxic effects of streptomycin on the eighth nerve on 21 men with tuberculosis. Each patient received 2 gm. of streptomycin daily for 121 days. All patients experienced vestibular dysfunction of varying degree, and only four now have normal responses to caloric tests. There was no loss of hearing noted. During treatment 17 had tinnitus.

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53. Winston, J.; Lewey, F. H.; Parenteau, A.; Marden, P. A., and Cramer, F.: Further Experimental Studies of the Toxic Effects of Streptomycin on the Central Vestibular Apparatus of the Cat, Ann. Otol. Rhin. & Laryng. **58**:987-996 (Dec.) 1949.

54. Berg, K.: The Toxic Effect of Streptomycin on the Eighth Cranial Nerve, Ann. Otol. Rhin. & Laryng. **58**:448-456 (June) 1949.

55. Wallner, L. J.: The Otologic Effects of Streptomycin Therapy, Ann. Otol. Rhin. & Laryng. **58**:111-116 (March) 1949.

56. Peik, D. J.; High, H., Jr., and Campbell, P.: Streptomycin Toxicity of the Eighth Nerve, Arch. Otolaryng. **50**:251-254 (Sept.) 1949.

Abstracts from Current Literature

Ear

A Clinical Classification of Vestibular Disorders with Differentiation of Three Syndromes and Discussion of a Common Form of Vertigo Induced by Sudden Movement of the Head. F. R. Ford, Bull. Johns Hopkins Hosp. **87**:299 (Oct.) 1950.

Vertigo is a common and distressing sypmtom, but there is no general agreement about the interpretation of even the types which occur most frequently. Ford separates the disorders of the vestibular apparatus into three chief syndromes: (*a*) those due to spontaneous discharge, (*b*) those due to increase of vestibular reflexes, and (*c*) those due to loss of vestibular reflexes.

The first syndrome, i. e., symptoms produced by spontaneous discharge, is observed in an isolated form in Ménière's disease. The same symptoms may occur in acute processes involving the labyrinth or the eighth nerve, such as labyrinthitis of various types, vascular disorders, intoxications, trauma, and perhaps allergic reactions. In cases of this type, the picture is complicated for the time by increase in reflexes and eventually by loss of reflexes.

The second syndrome, i. e., symptoms due to increase of vestibular reflexes, is seen not only in association with labyrinthitis and with various types of vestibular irritation, as mentioned above, but much more commonly in subjects of middle age who have been subjected to prolonged nervous strain. In such patients the symptoms are usually transient and there is no deafness or loss of function. This condition has been termed "postural vertigo," but since the vertigo is induced by movement of the head it might well be termed "movement vertigo." In elderly patients these symptoms are very common and are apt to be persistent.

The third syndrome, i. e., symptoms due to loss of vestibular function, may result from any destructive process involving the vestibular apparatus, whether acute or slowly progressive. When one labyrinth or one eighth nerve is involved, the symptoms are usually of only a few months' duration, for compensation eventually occurs; but when both nerves are involved, the symptoms are persistent. Ford points out that it is important to distinguish between discharge and loss of function, so as to avoid useless operations.

Alpers, Philadelphia [A. M. A. Arch. Neurol.].

Tuberculous Otitis and Meningitis. D. I. Arbuse and E. Levenson, J. Nerve. & Ment. Dis. **113**:165 (Feb.) 1951.

Of a series of 42 patients with tuberculous meningitis, two had associated tuberculous otitis media. One, a man aged 46, had chronic otologic disease of three years' duration, and one year prior to his admission the aural discharge was found to contain tubercle bacilli. He died of lymphocytic meningitis, from which tubercle bacilli were not cultured. The second patient, a girl aged 18 years, had had a draining ear for 10 years, from which tubercle bacilli were ultimately cultured. Lymphocytic meningitis developed, and pellicle and Levinson tests gave positive results. She showed improvement for one year while receiving streptomycin therapy.

Berlin, Chicago [A. M. A. Arch. Neurol.].

Toxic Effects of Streptomycin on the Eighth Nerve. Raul Velasco L., Rev. otorrinolaring. **10**:103 (Dec.) 1950.

Velasco has made a clinical study of 194 patients in the bronchopulmonary department of the Salvador Hospital. Of this series of tuberculous patients, 88 were eliminated from the study for evaluation of the toxic effect of streptomycin on the eighth nerve. The patients were grouped under three categories depending upon the dose administered: (*a*) those who received from 15 to 19 gm., (*b*) those who received 30 to 35 gm., and (*c*) those who received 45 to 51 gm. In analyzing the patients, examination of both cochlear and vestibular nerves was made prior to the institution of treatment and at regular intervals during the course of treatment. Velasco noted that while the auditory function in patients suffering from tuberculosis was within normal

limits, the vestibular function showed a hypoexcitability. The daily dose of streptomycin varied between 0.5 gm. and 3 gm. The patients receiving 0.5 gm. a day showed no vestibular symptoms. However, as the dose of streptomycin increased there was a definite increase in the disturbance as shown by the vestibular findings. Forty-two per cent of the patients received 1 gm. a day, 63% received 1.5 gm. a day, and 90% received 2 gm. a day. Once there were vestibular disturbances, regeneration of the vestibular function rarely occurred. Velasco also believes, from his clinical observation, that, whatever the lesion may be, it is probably a peripheral lesion and also that the patients with tuberculosis are more susceptible to toxic effects of streptomycin than normal patients. He also notes that there is a probability that the drug may produce toxic effects in the fetus.

PERSKY, Philadelphia.

Pharynx

THE TONSIL AFTER RADIUM IRRADIATION. ERNEST J. VAN EYCKEN, Ann. Otol. Rhin. & Laryng. **59**:648 (Sept.) 1950.

Van Eycken reports the histopathological observations on a patient upon whom tonsillectomy was performed 20 years after radium irradiation of the tonsils. The patient, a 52-year-old white woman, had been treated in 1928 by insertion of one radon seed into each tonsil for three days. It was estimated that she had received 166.8 mc.-hr. to each tonsil.

Clinical examination showed large, chronically inflamed, cryptic tonsils with injection of the anterior pillars and lingual tonsillitis. Numerous strong adhesions were encountered during tonsillectomy.

Histopathologically, the tonsils were well encapsulated, the surface epithelium showing mild hyperkeratosis. Moderate hyperplasia of the lymphoid elements was noted. Fibroplasia, with dense proliferation of scar tissue, was a prominent feature. Occasional islands of metaplastic hyaline cartilage were encountered in the fibrous tissue. Vessel walls appeared thicker than usual and showed narrowed lumina. Atrophy of the tonsils with probable irradiation sclerosis was the histopathological diagnosis.

STEELE, Kansas City, Kan.

USE OF HYALURONIDASE WITH LOCAL ANESTHETIC IN TONSILLECTOMY. C. J. HEINBERG, Eye, Ear, Nose & Throat Month. **30**:31 (Jan.) 1951.

Heinberg reports the use of hyaluronidase with local anesthesia in tonsillectomy. The hyaluronidase is used in a dose of 150 TR (turbidity reducing) units added to 25 cc. of 2% procaine solution combined with 5 cc. of epinephrine. Because of the more rapid and more widespread diffusion, the operative procedure is made more comfortable, the procedure may be started immediately after injection, and less of the injection solution is necessary. Healing is not retarded, and there were no untoward reactions, either local or general.

The preparation is stable indefinitely in the dry state and for about two weeks when in solution. Heinberg, however, has the solution prepared fresh for each procedure.

JENNES, Waterbury, Conn.

Larynx

THE TREATMENT OF LARYNGEAL TUBERCULOSIS WITH STREPTOMYCIN. TIBOR VON BAJKAY, Pract. oto-rhino-laryng. **13**:76, 1951.

Von Bajkay presents a series of eight cases of pulmonary tuberculosis complicated by frank pathological condition of the larynx and pharynx. The symptomatology in these cases is that of advanced involvement of these structures. Streptomycin therapy was instituted in daily doses of 0.5 to 3.0 gm. intramuscularly. The average dose was 0.2 gm. five times a day. After the fourth or fifth day, frank improvement was noted. Dysphagia disappeared, the temperature and cough subsided, the general condition improved, the appetite and body weight had increased, and the local pathological condition and ulceration disappeared rapidly. With oral administration of this drug no evidence of improvement was shown. However, von Bajkay cautions against too optimistic prognosis, since in a number of these cases relapses occur. He feels that streptomycin therapy is positively indicated in cases of frank disease of the larynx and pharynx.

PERSKY, Philadelphia.

INTUBATION AND TRACHEOTOMY IN THE TREATMENT OF LARYNGEAL DIPHTHERIA. S. CHAROCOPOS and M. KOLOKOURI, Arch. Greek paediat. Soc. **1**:72, 1947.

Charocopos and Kolokouri report a study of 1,137 cases of laryngeal diphtheria treated in the Children's Hospital (Athens) during the years 1922 to 1942.

After describing the pathogenesis, the clinical forms, and the factors influencing the incidence, they present in detail the methods of treatment used by them and by other workers and their results. They describe the advantages, the indications, and the complications of intubation and tracheotomy.

The following is a summary of their results. 1. The highest seasonal incidence of laryngeal diphtheria was noticed in November (203 cases), December (197 cases), and October (167 cases) and the lowest in July (25 cases) and June and August (33 cases in each). 2. The highest age incidence was noticed in the second (300 cases) and the third (244 cases) years of life. 3. There were 313 deaths, or a mortality of 27.4%. The cause of death was myocarditis in 44.4%, myocarditis and bronchopneumonia in 20%, pneumonia in 11.1%, and occlusion of the trachea in 6.6%. 4. The percentages for mortality in the cases in which neither intubation nor tracheotomy was performed were 12.8; in the cases with intubation, 35.5; in the cases with intubation followed by tracheotomy, 40, and in the cases with tracheotomy without preceding intubation, 50.

The treatment indicated in laryngeal diphtheria consists in administration of diphtheria serum as early as possible. If in spite of serotherapy the signs of dyspnea persist and become more marked so that the life of the child is in danger, intubation should be performed, and if this fails to relieve the dyspnea, then tracheotomy should be performed.

DOXIADIS, Newcastle, England [AM. J. DIS. CHILD.].

Nose

ACUTE SUPPURATIVE FRONTAL SINUSITIS: INTRACRANIAL COMPLICATIONS. J. H. MAXWELL, Ann. Otol. Rhin. & Laryng. **59**:451 (June) 1950.

The intracranial complications of suppurative frontal sinus disease of which the rhinologist must be aware are chiefly osteomyelitis of the skull, epidural abscess or hemorrhage, diffuse subdural abscess, thrombosis of the superior longitudinal sinus, cavernous sinus thrombosis, leptomeningitis, diffuse purulent encephalitis, and localized frontal lobe abscess.

The danger signs in suppurative sinusitis are spread of infection beyond the anatomical boundaries of the sinus. They include external swelling of the forehead, glabella, and eyelids, generalized headache, vomiting, sensorial change, visual disturbance, cranial nerve palsy, convulsions, chills and spiking fever, disproportion between signs and symptoms, acute increase in intracranial pressure, and high leucocyte count (above 20,000 or 25,000 in an adult).

Osteomyelitis of the skull, secondary to frontal sinus suppuration, has become rare since the advent of antibiotic and chemotherapeutic agents. When established, osteomyelitis of the skull demands radical excision of the affected bone. Epidural abscess requires wide removal of the skull in the region of the infection until healthy dura is exposed. Diffuse subdural abscess, in almost every instance, is a complication of an acute fulminating sinusitis rarely developing during an acute exacerbation of a chronic infection.

Thrombosis of the superior longitudinal sinus is very rare, perhaps because of the anatomical structure of the sinus. This sinus has a heavy covering of dura and is extremely small where it is in close proximity to the frontal sinus. The decrease in morbidity and mortality of cavernous sinus thrombosis in recent years has been made possible by advances in chemotherapy and the antibiotic agents. Leptomeningitis may occur early in the course of an acute suppurative sinus disease as the result of septic thrombophlebitis of the communicating veins, or it may be the late result of a slower spread of infection by contiguity of tissue.

In considering frontal lobe brain abscess, one should remember that such a lesion may be present in the patient who remains ill with clinical evidence of undrained pus after the sinus infection is under control as the result of medical or surgical treatment. Differential diagnosis from clinical examination alone among frontal lobe abscess, subdural abscess, and even epidural abscess may be impossible. There is much less clinical evidence of an infectious process in

epidural abscess than in subdural or brain abscess. Diagnostic trephination of the skull, ventriculography, and exploratory tapping may be required before a positive diagnosis can be made.

If there is clear-cut evidence of a frontal lobe abscess, it should be attacked first by tapping and finally by excision. Later, a complete external frontal and ethmoid sinus exenteration, including removal of the posterior frontal wall, must be performed. If there is evidence of intracranial spread of infection but the exact nature of this is not known, the sinus surgery should be done immediately. The posterior wall of the frontal sinus must be removed to permit a thorough study of the underlying dura. The only acutely infected frontal or ethmoid sinus that should be operated upon is the one with evidence of extension of infection to the calvarium or intracranial contents.

STEELE, Kansas City, Kan.

NASOPHARYNGEAL TUMORS: REPORT OF OBSERVATIONS AT THE NIIGATA UNIVERSITY OTOLARYNGOLOGICAL DEPARTMENT. YOSHIDA OGINO, Jap. J. Otolaryng. **23**:135 (April) 1951.

Ogino reports his clinical observations on 38 patients with benign and malignant tumors of the nasopharynx. They were observed in 15 years at Niigata University Medical Hospital. All the 10 cases of nasopharyngeal fibromas occurred in males under 20 years of age. Their chief complaints were nasal obstruction and epistaxis. At the time of admission these symptoms had existed for one to six years. The fibromas responded well to surgery and radium irradiation. Of the 28 cases of malignant neoplasm in the nasopharynx, 11 were classified as sarcoma, 7 as carcinoma, 1 each as endothelioma and lymphepithelioma, and 8 were histologically undetermined. The chief complaints of patients with carcinoma were headache, impaired hearing, tinnitus, and paralysis of the fifth, sixth, and seventh cranial nerves. Cervical metastasis was not observed in all patients on admission to the hospital but appeared later in the course of the disease. The duration of the subjective symptoms was one to eight months at the time of admission to the hospital. Every patient was male and was past 40 years of age. In cases of sarcoma, nasal obstruction and cervical adenitis were the chief symptoms which brought the patients to the clinic. The nasopharyngeal fibromas were extirpated through the nose in seven instances and through the oral route in two. Two patients died in the hospital. Six patients with carcinoma received combined treatment of surgery and irradiation. One was treated with irradiation only. Seven patients with sarcoma received radium irradiation, and four were treated by surgery and irradiation. Nasopharyngeal fibromas had the best prognosis. Ten have survived for a four-year period among the patients with malignant growths.

HARA, Los Angeles.

Miscellaneous

ACUTE SUPPURATIVE INFECTIONS OF THE SALIVARY GLANDS IN THE NEWBORN. BERNARD H. SCHULMAN, Am. J. Dis. Child. **80**:413 (Sept.) 1950.

Schulman says that sialadenitis is not an uncommon infection in the newborn infant, especially in the parotid gland, with the submaxillary gland next in frequency. Two cases are reported. The first was of a white girl delivered by Cesarean section who weighed 6 lb. 4 oz. (2,835 gm.) and was placed immediately in an incubator and bottle fed. The seventh day her temperature was 102.4 F., and there was a large tense swelling of the left parotid gland. Pus could be expressed from Stensen's duct. A culture showed Staphylococcus aureus. Penicillin, 50,000 units, was injected intramuscularly, followed by 25,000 units every three hours. The red blood cell count was 5,020,000, the white blood cell count 23,200, with 41% polymorphonuclear leucocytes and 6% eosinophils. The temperature was normal three days later, and the patient was dismissed in one week. The second patient was a 3-week-old Negro boy with a right-sided swelling of the jaw which exuded pus on pressure. There was no maternal history of sepsis. A culture showed Staph. aureus. Penicillin was given intramuscularly, 20,000 units every three hours. The temperature was normal in two days, and release from the hospital occurred on the eighth day.

Infection takes place either by transmission from the mouth through the salivary duct, or by way of the blood from a metastasis. It is suggested that removal of mucus from the mouth by a gauze-covered finger may cause trauma of the duct orifice with resultant infection. The

germ is either a Streptococcus or a Staphylococcus but may be also Escherichia coli. The differential diagnosis is mumps or hemangioma of the parotid or, of course, cervical adenitis. Surgery is seldom necessary since the advent of penicillin and the sulfonamides.

VOORHEES, New York.

RESPIRATORY ACIDOSIS IN CHILDREN WITH CEREBRAL PULMONARY AND CARDIOVASCULAR DISORDERS. DOUGLAS E. JOHNSTONE and ERIKA BRUCK, Am. J. Dis. Child. **80**:578 (Oct.) 1950.

There are few case reports such as these in the literature. Perhaps many are overlooked. Johnstone and Bruck discuss the following: A. conditions in which there is suppression of respiration, (1) damage to central nervous system, (2) loss of "ventilatory function" in the lung, (3) effect of pain (pleurisy); B. conditions characterized by impairment of diffusion of carbon dioxide across the alveolar membrane; C. conditions in which there is an obstacle to the escape of carbon dioxide from the alveoli; D. conditions in which the pulmonary blood flow is insufficient.

Sixteen cases of respiratory acidosis are presented with clinical and laboratory data and a classification of causation. In five cases respiration was suppressed through cerebral dysfunction. In seven cases the pH of the blood serum was below 7.0 and in four others there was metabolic acidosis. With respiratory anoxia based on acidosis there is cyanosis. With oxygen tension in the arteries of over 70 mm. of mercury, the cartoid sinus and aortic mechanisms will not maintain respiration. However, practically no ill effects from oxygen administration have been noticed in this study. The condition has some significance in administration of gaseous anesthetics, for example in removal of adenoids and tonsils in children.

VOORHEES, New York.

THE REMOVAL OF AIR FROM THE RESPIRATORY TRACT AND CERTAIN OTHER BODY SPACES UNDER NORMAL AND ABNORMAL CONDITIONS. A. C. HILDING, Ann. Otol. Rhin. & Laryng. **59**:309 (June) 1950.

The absorption of air from the anterior chamber of the eye, from the sinuses, from the ear, and from portions of the lung distal to obstruction follows the principles of well-known laws of physics governing diffusion, solubility, partial pressures, and molecular combination.

It has been shown that air can be absorbed completely from almost any space or tissue in the body, such as those mentioned, as well as the peritoneum, pleural cavity, or subcutaneous tissues, provided sufficient time is allowed and provided the space containing the air can collapse. If the space cannot collapse or can do so only partially, negative pressure develops, depending on the gas pressure present in the absorbing fluid.

In postoperative atelectasis, there are at least three mechanisms by which the air is removed, namely, (1) absorption, (2) displacement by secretion, and (3) the pumping or piston action of the cilia and moving masses of mucus. The negative pressure in most cases of postoperative atelectasis is probably maintained solely by ciliary action. The piston-like action of the cilia and moving masses of mucus is a likely cause of vacuum sinus headache and negative pressure in the middle ear following tubal obstruction.

STEELE, Kansas City, Kan.

THE PRODUCTION OF NEGATIVE PRESSURES BY RESPIRATORY CILIA. JOHN J. BALLENGER, Ann. Otol. Rhin. & Laryng. **59**:694 (Sept.) 1950.

Ballenger attempted to corroborate previous experimental work by A. C. Hilding (*Ann. Otol. Rhin. & Laryng.* **52**:816-833, 1943), who reported negative pressure developed in a ciliated mucous membrane-lined cavity by the action of the cilia.

Utilizing methods as identical as possible to those of Hilding, Ballenger was not able to record negative pressures over 5 mm. of water due to ciliary action on a diaphragm of mucus in excised tracheas of rabbits, whereas the earlier author had reported readings of—35 to —36 mm. of water. Likewise, in experiments on negative pressure developed in the frontal sinus by ciliary action, Ballenger found an average of —15.5 mm. of water as compared with —66 mm. of water, the highest reading reported by Hilding.

STEELE, Kansas City, Kan.

BACTERIAL MENINGITIS: RESULTS OF TREATMENT IN SEVENTEEN CASES WITH A NEW SULFONAMIDE (GANTRISIN®). P. S. RHOADS, F. A. SVEC, and J. H. ROHR, Arch. Int. Med. **85**:259 (Feb.) 1950.

Ten patients with meningococcic meningitis were treated with gantrisin,® a new sulfonamide with the formula 3,4-dimethyl-5-sulfanilamide-isoxazole, which was used as the sole bacteriostatic agent. All recovered without complications resulting from the disease or the treatment.

Two patients with meningococcic meningitis, three with pneumococcic meningitis, and two with hemophilus influenzae meningitis were treated with gantrisin® and one of the antibiotics. One of these patients, shown at autopsy to have pneumococcic endocarditis, died. All the others recovered.

Gantrisin® was administered orally, intramuscularly, and intravenously, with no untoward effects. Several patients who had albuminuria and microscopic hematuria at the start of treatment with gantrisin® were given the drug in full doses without adjuvant alkali therapy; the conditions cleared up under treatment.

ALPERS, Philadelphia [ARCH. NEUROL. & PSYCHIAT.].

CAUSTIC STRICTURES OF THE ESOPHAGUS. PAUL H. HOLINGER and KENNETH C. JOHNSTON, Illinois M. J. **98**:246 (Oct.) 1950.

An analysis of 96 consecutive cases of caustic burn of the esophagus is presented. An analysis of the types of caustic agent ingested, the age groups, sex, and race incidence is given. Most cases occur in children and are accidental, the caustic being placed in a container ordinarily used for food or drink, such as a glass, cup, or soft drink bottle. Acute burns constituted approximately one-third of the cases, while in the remaining two-thirds the patients were first examined only after multiple strictures had developed. Most significant is the recent change in the management of the acute burn, dilatation within 24 hours of the accident by means of soft rubber mercury-filled bougies being started. No strictures have developed in the patients so treated during the three years this treatment has been used, and no complications, such as perforation, have developed. Once a caustic has been swallowed, dilatation or some form of esophageal examination must be continued throughout the life of the patient at increasing intervals. Failure to continue management when swallowing function is restored, either initially or in later years, results in cicatricial contracture of the severest type for which gastrostomy, multiple dilatations, or esophageal resection may have to be done.

FROM THE AUTHORS' SUMMARY [A. M. A. J. DIS. CHILD.].

STREPTOMYCIN IN THE TREATMENT OF INFLUENZAL MENINGITIS: A STUDY OF NINETY CASES, WITH 96.6 PER CENT RECOVERY. E. APPELBAUM and J. NELSON, J. A. M. A. **143**:715 (June 24) 1950.

In 90 consecutive cases of meningitis due to Hemophilus influenzae, streptomycin was given intramuscularly and intrathecally in 73 and intramuscularly only in 17. In addition, sulfadiazine was used in all but one, the specific rabbit serum in 17, and aureomycin in one. Of the 90 patients, 87 recovered and 3 died, a recovery rate of 96.6. The 17 patients treated with intramuscular, but without intraspinal, injections of streptomycin recovered completely.

With regard to adjunctive therapy, it was difficult to evaluate the role of sulfadiazine. There was no evidence that rabbit serum played a contributory role.

There were relatively few toxic reactions in the series, and neurological residua occurred in a small number of cases.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL.].

OTOLOGIC APPLICATIONS OF AUDIOLOGY. R. E. JORDAN, J. Speech & Hearing Disorders **16**:115 (June) 1951.

At the University of Pittsburgh Eye, Ear, Nose, and Throat Hospital, otology and audiology have been fitted together as a team for work on hearing problems both for diagnosis and for treatment. Three illustrative cases are presented showing the results of the cooperation of the two professions.

PALMER, Wichita, Kan.

A Survey of Services for the Speech and Hearing Handicapped in New England. Wilbert Pronovost, J. Speech & Hearing Disorders **16**:148 (June) 1951.

Seven and eight-tenths per cent of 87,288 persons tested were handicapped in speech in the New England area. The number of persons receiving remedial services was about 2% of the estimated number with speech and hearing handicaps in New England.

Palmer, Wichita, Kan.

Cerebral Palsy: Professional Training Activities in This Country. Martin F. Palmer, J. Speech & Hearing Disorders **16**:157 (July) 1951.

Only a few professional training schools in the field of speech disorders are offering separate academic material in the speech disorders of cerebral palsy. A number of other professional training institutions offer incidental lecture material.

Palmer, Wichita, Kan.

Infant Speech: Consonantal Position. Orvis C. Irwin, J. Speech & Hearing Disorders **16**:159 (June) 1951.

During the first two and one-half years of life the course of development of initial consonants is linear, that of the medials is decelerating, and that of the finals is accelerating. Final consonants are infrequent in the speech patterns during the first half year of life, while initial consonants are more frequent than medials.

Palmer, Wichita, Kan.

A Device for Continuously Variable Time Delay of Headset Monitoring During Magnetic Recording of Speech. Grant Fairbanks and Robert Jaeger, J. Speech & Hearing Disorders **16**:162 (June) 1951.

A description is given of an apparatus for introducing time delays into the auditory feedback circuits of human speech.

Palmer, Wichita, Kan.

Embryonic Hearing Organs After Maternal Rubella. LeRoy A. Schall, M. H. Lurie, and G. Kelemen, Laryngoscope **61**:99 (Feb.) 1951.

The incidence of deafness in the progeny after the mother has had rubella during pregnancy is not definitely established. However, almost all such progeny have some kind of congenital defect if the rubella was contracted early in pregnancy.

There have been few pathological investigations. The authors present reports of two cases with pathological studies of the temporal bones. Hemorrhage and hyperemia were conspicuous. Other authors, too, have reported hemorrhage. It is difficult to tell how much is due to trauma and how much to rubella. The embryonic development of the organs was normal. This is to be contrasted with Carruthers' case and the organs of the waltzing guinea pig.

Rubella may cause deafness by two methods: (1) an arrest in the development of the organ of Corti and (2) vascular changes. The latter may possibly be causative of the former.

Hitschler, Philadelphia.

Headache and Autonomic Imbalance. Raymond L. Hilsinger, Laryngoscope **61**:296 (April) 1951.

Bellergal® (a combination of ergotamine tartrate, belladonna alkaloids, and phenobarbital) was used successfully in the treatment of migraine. It is a sympathetic and parasympathetic inhibitor and a sedative, which actions tend to increase the interval between attacks. Case reports are included in the article.

Hitschler, Philadelphia.

Observations in Treating Seven Cases of Choanal Atresia by the Transpalatine Approach. Harold Owens, Laryngoscope **61**:304 (April) 1951.

Owens uses a transpalatine approach which is a slight modification of Ruddy's method. This method is to be preferred over the blind intranasal removal of the obstruction for many reasons,

among which are these: The work is done under direct vision, the choana does not close postoperatively, and there is comparatively little postoperative care needed. The operation is described in detail and well discussed.

HITSCHLER, Philadelphia.

STUDY OF CORICIDIN IN THE TREATMENT OF THE COMMON COLD. MICHAEL O. KOVALEFF, New York J. Med. **50**:2955 (Dec. 15) 1950.

The Allergy Clinic of St. Luke's Hospital offered the background for this study. The antihistamine used was chlorprophenpyridamine (chlortrimeton) maleate combined in a tablet with acetylsalicylic acid, acetophenetidin (phenacetin®), and caffeine ("APC"). Three tablets were prescribed. The first contained the maleate just mentioned, 2 mg.; acetylsalicylic acid, 0.23 gm.; acetophenetidin, 0.15 gm., and caffeine, 0.03 gm. The second "APC" tablet contained acteylsalicylic acid, 0.23 gm.; acetophenetidin, 0.15 gm., and caffeine, 0.03 gm. The third tablet was a placebo. Identical in appearance, the tablets were administered to "the employees of a large insurance company in New York" for the treatment of upper respiratory infections as soon as possible after the initial signs developed. Ten tablets were given to each person, who was told to take two at once, then one every four hours while awake until the symptoms cleared. This was "a new cold tablet." A "study" was not mentioned. No other medication of any kind was allowed. After 48 hours, the subjects were questioned as to the number of tablets taken and their observation of any signs or symptoms, favorable or otherwise. "There was a high percentage of cures with placebo." Some persons may have misinterpreted their symptoms. Two tables are given in the article to show reactions and results. In comparison of the first medication with the second, the coricidin® (chlorprophenpyridamine maleate plus an acetylsalicylic acid-acetophenetidin, caffeine formula) seemed to give more relief than the APC tablets or the placebo. Coricidin® seems to have relieved 78.4% of the subjects, compared with 68.4% relief of those who took APC tablets.

VOORHEES, New York.

EVALUATION OF AUREOMYCIN, CHLOROMYCETIN AND OTHER NEWER ANTIBIOTICS. SAMUEL KARELITZ, New York J. Med. **51**:234 (Jan. 15) 1951.

Data from Mount Sinai Hospital and Willard Parker Hospital were used in making this comparative study of chloramphenicol (chloromycetin®), aureomycin, polymyxin, bacitracin, and terramycin. Polymyxin has a pejorative action on kidneys, but "should be used in clinically severe Bacillus pyocyaneus infections." It may be tried for three to four days in conditions resistant to other antibiotics, but its use may be "precarious." Terramycin (Chas. Pfizer & Company, Inc.) is similar in action to aureomycin but should not be used alone in influenzal meningitis. Bacitracin is synergistic with penicillin in syphilis, but in general it is not to be used parenterally. It is safe for skin and certain ocular conditions. Chloramphenicol, aureomycin, and terramycin were introduced after penicillin and streptomycin. All three are potent against rickettsiae, brucellosis, Hemophilus influenzae, and H. pertussis. Chloramphenicol is useful in typhoid and Salmonella infections. Aureomycin is potent against Gram-positive cocci. A choice in a given instance depends upon a patient's tolerance, the method of administration, the dosage required, and the cost. Two tables are given to show relative action of all these antibiotics, first for Gram-positive, second for Gram-negative, organisms. In summary, we have yet much to learn about antibiotics. Still more information will be required when better and more useful ones are developed. We are still rather helpless in the presence of virus infections.

VOORHEES, New York.

HEADACHES AND THEIR TREATMENT. GEORGE S. KING, New York J. Med. **51**:255 (Jan. 15) 1951.

King follows Wolff's dictum that migraine headaches are often due to changes in the cerebral blood vessels which may be classified as (1) the vasoconstrictor phase, (2) the vasodilator phase, and (3) the edema phase. Diagnosis of the cause of headaches has always been a troublesome problem. We must proceed with treatment after a careful diagnosis has been established, and this may require several sessions. Description of the attack must be meticulously set down. Emotional conflicts may be causative.

Classic migraine has three phases. Migraine variants have one or two of these phases, seldom three. Tension headache is definitely related to stress situations. It is slow in coming and less severe than are other types. Vasoconstrictors relieve vascular headaches. One may use a histamine test to differentiate between normal persons and persons with migraine. Ergotamine tartrate is almost a specific and has now become available in a tablet combined with caffeine containing 1 mg. of ergotamine tartrate plus 100 mg. of caffeine. Two such tablets are to be taken at the first sign of an attack and then one every half hour as necessary until six are swallowed in all. Parenteral or rectal administration may be advisable. If the physician has the patient's confidence, relief may be obtained through conversation and suitable advice. Diet and daily activities must be established in a routine to suit present needs. Coffee intake may require adjustment as to amount and frequency. It is assumed that some 8 million persons (in the United States) suffer from headaches, and in many instances they receive acetylsalicylic acid or "APC" medication only. This is usually futile.

VOORHEES, New York.

FURTHER STUDIES ON THE TREATMENT OF THE COMMON COLD WITH ANTIHISTAMINIC DRUGS. CHARLES R. SHAW and HENRY B. WIGHTMAN, New York J. Med. **51**:387 (Feb. 1) 1951.

These studies are a continuation of the studies reported from the Cornell University school of medicine. During 1950 the first report was made on 443 college students using a complicated organic benzyl-phenyl hydrochloride known as "A," exhibited for three days in 50 mg. doses. About half the subjects received the drug; the other half received a placebo only. "It was concluded that the antihistaminic drug was of no benefit in treating the colds." In this report a "B" formula was used, a stronger antihistamine than "A" but about equally active. The dosage was 15 mg. A third compound known as "C" was used also for two months (April and May). "This is one of the most popular over-the-counter antihistamines sold." A placebo equal to that in the A and B tests was administered as a control. Five tables in this article set down the results against sore throat, congestive nose, cough, and malaise. In all, 911 subjects were studied, half receiving a placebo. "No definite trend in improvement is evident in the treated cases as compared with those treated by an inert placebo or with aspirin." The authors further observe, "The beneficial effects of treating the common cold with an antihistaminic drug appear questionable."

[Abstractor's Comment: Paul De Kruif and the widely read *Reader's Digest* please take notice.]

VOORHEES, New York.

COMPRESSION OF THE TRACHEA OR ESOPHAGUS BY VASCULAR ANOMALIES: SURGICAL THERAPY IN 40 CASES. ROBERT E. GROSS and EDWARD B. D. NEUHAUSER, Pediatrics **7**:69 (Jan.) 1951.

Description is given of five different types of anomalies of the great vessels in the superior mediastinum which can produce compression of the esophagus or trachea (or both). These malformations of the vascular system can give rise to difficulties in swallowing and to serious disturbances in pulmonary ventilation. These anomalies include double aortic arch, right aortic arch with a left ligamentum arteriosum, anomalous innominate artery, anomalous left common carotid artery, and an aberrant subclavian artery.

The various symptom complexes which can be found with these anomalies are described. Endoscopic visualization of the interior of the esophagus or trachea gives valuable information in some areas. The investigation of these subjects should include fluoroscopic and roentgenologic studies of the esophagus and trachea by contrast medium. Data gathered from such roentgenologic examinations usually give a fairly clear idea of the type of vascular anomaly which is present.

It is possible to attack surgically each of these vascular malformations. In general, the esophagus or trachea can be relieved by division of an anomalous vessel or by displacement of an artery in such a manner that it is carried away from the compressed structure. When necessary, these operative procedures can be undertaken in very young babies, even in the presence of a high degree of respiratory obstruction. All the operations have been through a left anterolateral, transpleural approach, with the patient under general anesthesia with a closed system. The results of operation in 40 patients are analyzed.

It is evident that many vascular anomalies in the thorax which disturb the functions of the esophagus or trachea can now be treated with an excellent chance of relieving the obstructive symptoms.

FROM THE AUTHORS' SUMMARY [A. M. A. J. DIS. CHILD.].

MENINGIOMAS OF THE CEREBELLOPONTINE ANGLE. G. BAKER and L. A. CHRISTOFERSON, Proc. Staff Meet., Mayo Clin. **25**:549 (Sept. 13) 1950.

Although meningiomas are usually benign tumors, the proximity of vital centers within the brain stem and pons makes their complete removal from the cerebellopontine angle extremely difficult and hazardous. In addition, in order to effect removal from this location, it is usually necessary to sacrifice one or more cranial nerves. The case here reported, that of a woman aged 44, was an exception.

Diagnostically, the patient presented an interesting situation because of the emphasis placed on dysphagia and dysarthria and because she did not suffer from headache and presented no signs of involvement of the fifth cranial nerve. Therapeutically her case was even more interesting, not only because the tumor was removed without visible damage to the brain or cranial nerves but also because the function of the involved cranial nerves was improved. The left acoustic nerve had not functioned for over two years, and yet the hearing returned completely after total removal of the meningioma.

ALPERS, Philadelphia [A. M. A. ARCH. NEUROL.].

H. INFLUENZAE MENINGITIS. I. A. B. CATHIE and S. D. v. WELLER, Arch. Dis. Childhood **23**:208 (Sept.) 1948.

A case of Hemophilus influenzae meningitis is presented in detail. Treatment with streptomycin was a total failure, and the disease was only partly controlled by use of penicillin and sulfonamide compounds. After three relapses, the disease was cured by the additional administration of H. influenzae rabbit antiserum.

KELLY, Milwaukee [AM. J. DIS. CHILD.].

CHRONIC MAXILLARY SINUSITIS IN THE INFANT. MASPÉTIOL, CHAUVET, and CARRÉ, Ann. oto.-laryng. **67**:758 (Nov.-Dec.) 1950.

Contrary to the common impression and general belief, isolated chronic sinusitis without ethmoidal involvements is relatively frequent in the infant. In fact, it is more frequent in the infant than is ethmoiditis.

A study of 202 roentgenograms demonstrated the following findings: (1) roentgenologic evidence of involvements of the maxillary sinus in 114 patients without evident ethmoidal infection, (2) ethmomaxillary involvement in 12, and (3) ethmoidal opacity alone in 6. Diagnostic punctures in 41 patients revealed positive findings in 28.

Chronic maxillary sinusitis is often overlooked because of the fact that the functional rhinopharyngeal signs are not striking. The usual rhinoscopic signs of sinusitis are generally absent. A definite diagnosis can be obtained only by routine and systematic roentgenograms and antral punctures when indicated.

VIOLE, Los Angeles.

DEVELOPMENTAL ANOMALIES OF THE ESOPHAGUS. JOSEFA LADANYI and SANDOR SZUCS, Ann. pædiat. **173**:1 (July) 1949.

In the teratogenic explanation of esophageal atresia, the mechanical theory, as presented by Happich, Keith, Zausch, and J. A. Schmitz, is recently coming more to the fore. The authors agree with this explanation, because in the three cases of esophageal atresia in combination with esophagotracheal fistula observed by them there was further found in each case an open foramen ovale; in addition, in one case an open ductus arteriosus was found; in another, atresia ani vaginalis, and in the third, a stenosed rectum. These findings cannot in any way be explained by the occlusion theory brought forward formerly by Tandler and Kreuter, but they appear much more to be caused by pressure, which may be common in all cases.

From a review of the development of the operative technique it can be seen that the most complex problem is not to have the patient get over the difficulty of swallowing but to mend the fistula coexistent with the atresia that even after gastrostomy may lead to regurgitation of food and to its aspiration.

FROM THE AUTHORS' SUMMARY [A. M. A. J. DIS. CHILD.].

THE PATHOGENESIS AND TREATMENT OF HABIT VOMITING IN INFANTS. V. BAFFI, Pediatria **57**:373 (May) 1949.

Five cases are reported of infants in whom an almost intractible habit vomiting was associated with an enlarged thymus gland. X-ray therapy and the giving of cortical extract proved to be efficacious.

HIGGINS, Boston [A. M. A. J. DIS. CHILD.].

FOREIGN BODIES IN THE ESOPHAGUS WITH SYMPTOMS EXCLUSIVELY FROM THE AIR PASSAGES. H. F. FABRITIUS, Acta pædiat. **37**:335, 1949.

Fabritius reports the cases of two children in whom large foreign bodies remained undetected in the esophagus for two and one-half and three months respectively, despite repeated examinations. He points out that neither of the patients had noticeable difficulties in swallowing but that they presented almost exclusively symptoms from the respiratory tract. The symptoms were very confusing and thus involved diagnostic difficulties. He discusses the two cases and, among other things, emphasizes the necessity of roentgenographic studies with contrast medium even for small children.

FROM THE AUTHOR'S SUMMARY [A. M. A. J. DIS. CHILD.].

FERROUS CARBONATE IN TREATMENT OF TIC DOULOUREUX. L. M. DAVIDOFF and E. H. FEIRING, Acta psychiat. et neurol. **24**:403 1949.

Davidoff and Feiring review the literature on the use of ferrous carbonate in the treatment of trigeminal neuralgia. B. Hutchinson ("Cases of Neuralgia Spasmodica, Commonly Termed Tic Douloureux, Successfully Treated," Ed. 2 London, Longman [and others], 1822), appeared to have been enthusiastic about this form of therapy. The present authors evaluated this method in the treatment of 91 patients—49 men and 42 women, between the ages 23 and 79, with 85% of all patients in the age range of 41 to 20 years. The average duration of illness was five years. The second and third branches of the fifth nerve were most frequently involved. Of the 91 patients, 57 reported no relief of symptoms; 34 patients, or about one-third, benefited appreciably. At the time of the report, improvement had lasted from six months to six years. Recurrence of pain was noted in 17 patients, and operation was undertaken on 11 of these.

The treatment consists in the administration of 4 capsules of ferrous carbonate, each containing 1 gm., twice daily with meals. A total of 8 gm. daily is used, and the treatment is continued from weeks to months. Improvement appears one to two weeks after therapy is instituted. Side reactions to the medication include nausea, vomiting, diarrhea, and constipation and may be countered with elimination of the drug for several days.

J. E. PISETSKY, New York [A. M. A. ARCH. NEUROL.].

Society Transactions

AMERICAN LARYNGOLOGICAL ASSOCIATION

Gordon B. New, M.D., *President*

Seventy-Second Annual Meeting, Atlantic City, May 9-10, 1951

Abstracted by Samuel Salinger, M.D.

Advances in Otolaryngology: Address of the President. Dr. Gordon B. New, Rochester, Minn.

In his address Dr. New pointed out the advances in otolaryngology during the past year which were the results of contributions from other fields of medicine. From the field of microbiology, the diseases of histoplasmosis, coccidioidomycosis, and actinomycosis as affecting the ear, nose, and throat, came to our notice, with the result that clinical advances were effected in their management. From pathology we learned to grade malignant tumors, gained an appreciation of the value of frozen sections, and were made aware of the newer cytologic procedures in diagnosis. Hematology contributed a knowledge of blood dyscrasias which are important in pre- and postoperative management; also to hematology we owe blood banks, the grouping of blood donors, the value of plasma, and many other valuable items so essential in the management of surgical cases. Chemotherapy added to our armament in the treatment of both medical and surgical patients, as have the various antibiotics, all of which have not only controlled various types of infection but have added to the safety of surgical procedures. Radiology has given us valuable therapeutic aids against both benign and malignant lesions as well as the surgical diathermy widely employed in place of cold dissection. New methods of anesthesia have added to the safety of many surgical procedures, contributing as well to the comfort of the patient and reducing postoperative sequelae.

Thus one learns the lesson that progress in a specialty comes not alone from refinements within but from the adoption of ideas developed in other fields and that one must be constantly on the alert for ideas which, though seemingly unrelated in the beginning, may eventually be converted to useful purpose in the fields of our particular endeavors.

Viral Infections of the Nose and Throat. Dr. John J. Shea, Memphis.

Since the advent of the antibiotics, which have been so effective in the control of bacterial infections, interest has shifted to the viral infections because of their resistance to these agents. Among the virus infections which to date have failed to respond are mentioned the common cold, poliomyelitis, virus pneumonia, herpes, mononucleosis, and influenza. Shea discussed the affinity of certain viruses for certain types of tissue and detailed the present status of our knowledge of virus. Specifically, he discussed in detail mononucleosis, mumps and the Stevens-Johnson syndrome of stomatitis, skin eruption, ulceromembranous lesions of the mucous membranes, and ophthalmia. Antibiotics have proved of little value because the virus, finding its habitat within the cell itself, is protected from the action of the drug. He believes that future therapy will probably be centered around the development of a vaccine such as has proved effective in certain types of influenzal infections.

DISCUSSION

Dr. Henry L. Williams, Rochester, Minn., stated that the use of antibiotics has decreased the incidence of suppurative and bacterial infections, thus bringing into prominence the virus infections which are resistant to most antibiotics. Diagnosis is often difficult in the early stages, especially the differentiation between acute tonsillitis and mononucleosis. Williams has not found nicotinic acid as effective against the latter as has been maintained by some. Terramycin seems effective in some cases but cannot be accurately appraised because of wide variability in the

manifestations of the diseases and because of the conflicting effects of penicillin and other antibiotics given at the same time. However, Dr. Williams has had success with terramycin in the treatment of colds complicated by herpes labialis.

In virus conditions, as in dermatologic affections, the lack of adequate information as to the true nature of the physiologic mechanisms involved leads to diversified nomenclature and unstable bases upon which to assess the results of therapy.

Dr. Henry M. Goodyear, Cincinnati, advised intravenous injections of pyridoxine hydrochloride, 125 to 150 mg., in cases of acute infectious agranulocytosis. It acts promptly in reducing temperature and improving the patient's general condition. In acute infectious mononucleosis he has found folic acid very effective when given hypodermically in 10-mg. doses three times daily.

Dr. W. W. Wilkerson, Nashville, Tenn., stated that viruses show very definite characteristics: first, a specificity for certain tissues, and second, when transmitted from one host to a different type of host, a rapid transition in the type of the disease, as, for example, yellow fever in man when transmitted to mice produces encephalitis. There is great doubt as to whether the virus is a living organism, many believing that it is a fabrication of the tissue cell itself. Also, it is not certain whether the virus causes an inflammatory reaction or whether the latter is due to secondary invaders. The bizarre symptoms of viral diseases are explained by the prolonged activity of the virus within the cell itself. Normal cell activity is disorganized, leading to certain vitamin and hormonal imbalances. The action of antibiotics in this connection is confusing, since it is known that these agents are assumed to exert their influence only in the fluids of the tissues surrounding the cells. Yet certain of them have been effective against viruses. The question is, do these viruses migrate into the inflammatory extracellular spaces? Furthermore, it can be argued that penicillin and streptomycin are distinctly contraindicated, because in the culture media of the fungus Histoplasma they actually produce a more luxuriant growth.

Dr. John J. Shea, Memphis, in closing, replied to a query as to the rationale of nicotinic acid therapy that the agent is effective against other forms of sore throat aside from the virus types, because it relieves pain.

Treatment of Osteomyelitis of the Skull Resulting from Ethmoidal and Frontal Sinuses. Dr. W. Likely Simpson, Memphis.

Dr. Simpson's paper dealt chiefly with the therapy of osteomyelitis. In acute cases with severe pain and external swelling he advised external drainage and adequate antibiotic therapy. Pictures were shown of a rubber tube in place in the ethmoid sinus of a child. Symptoms of osteomyelitis, such as edema, tenderness, swelling, and fever, are present before the roentgenograms reveal bony changes. The author advocates wide exposure and removal of all diseased bone via a supraorbital incision, which may be bilateral and divided by a midline vertical incision, creating two flaps. After the process has been brought under control it may be necessary to do a secondary operation in which the ethmoid sinuses are thoroughly exenterated. The original frontal wounds left open for drainage may be closed at a later date, and at even a later period a cosmetic procedure may be required to correct the deformity. Bone implants and tantalum plates have been successful in various types of cases.

DISCUSSION

Dr. Dean M. Lierle, Iowa City, expressed his preference for medullary bone obtained from the iliac crest for the correction of the postoperative defects. He finds it convenient to scoop the bone with a curette and stuff the fine bony masses into a previously prepared pocket under the skin covering the defect. This makes for a smoother filling than can be obtained with bone chips and is less laborious than the shaping of a single bone implant.

Dr. G. B. New, Rochester, Minn., stated that he had used a shredder or grinder for reducing the bone graft to a soft pliable mass, which is easily molded into the prepared pocket.

Dr. Harry P. Schenck, Philadelphia, was of the opinion that the use of large doses of antibiotics would in many cases obviate the necessity for radical surgery, provided adequate simple drainage were effected early. Much depends on selection of the proper antibiotic for a

particular invader in the individual case. Unless intracranial complications seem to be impending, it is best to delay surgery until the x-ray pictures can accurately delineate the extent of the process and determine whether it has been arrested. Frequently, after the process has been arrested one may see evidences of early bone regeneration. Thus conservatism may yield results and forestall a major deforming procedure.

Dr. W. L. Simpson, Memphis, closed the discussion by stating that he did not have the grinder mentioned by Dr. New and had, therefore, to rely on bone chips. As for Dr. Schenck's remarks on the conservative attitude, he agreed that we must be conservative in all cases, relying on early drainage and adequate antibiotic therapy. However, one cannot rely too heavily on roentgen findings, since these may often be misleading and osteomyelitis may be present despite negative roentgen findings. Clinical evidence would then have to prevail if symptoms of spreading bone disease were present.

Cortical Venous Thrombosis: Its Location and Its Sources. Dr. E. A. Stuart (by invitation); Dr. F. H. O'Brien (by invitation), and Dr. W. J. McNally, Montreal, Canada.

Dr. Stuart reported 16 cases, three of which were confirmed at autopsy, in which thrombosis of cerebral cortex veins produced a characteristic group of symptoms: aphasia, focal seizures, and hemiplegia. In addition, there were other cranial symptoms suggesting brain abscess, which, however, was ruled out by either exploration or negative ventriculographic findings. In 5 of 10 cases that were followed up for some time, epilepsy was a lasting complaint. As a corollary to this finding, an analysis of 85 epileptics in an institution showed unmistakable evidence of a preceding febrile or toxic state.

The localization of the symptoms was explained in slides showing the cortical distribution of the various motor centers and the venous circulation in these areas. The motor areas for the face and hand occupy a larger area than those for the foot and leg. Circulation at the Sylvian fissure may be slow because of anastomosis in both directions (up and down). Thrombosis of the prefrontal veins by extension from the superior longitudinal sinus results in edema of the cortical areas, affecting motor function of the face and hand. A slide was shown depicting thrombosed veins in the cortex extending to the vein of Trolard, with arachnoidal hemorrhage. Differentiation of thrombosis from brain abscess or subdural abscess may be delayed or obscure in certain cases. The therapy suggested so far as surgery is concerned consisted of adequate removal of the primary focus in the sinuses or ear. Eight cases of nasal origin were cited in which the patients were operated on, with recovery in four.

DISCUSSION

Dr. James M. Robb, Detroit, stated that persistent blood examinations should eventually disclose the nature of the bacterial invader, which should be of considerable value in the therapy.

Dr. Samuel Salinger, Chicago, stressed the importance of differential diagnosis of thrombosis and subdural abscess, which produces a group of symptoms similar to those described by the authors. It is important because the therapy of the two conditions is different. Subdural abscess requires adequate wide exposure and drainage, whereas surgery of thrombosis of the cortical veins is confined to eradication of the initial focus.

Dr. H. L. Williams, Rochester, Minn., believed that the authors had limited the symptomatology too severely in associating it solely with cortical venous thrombosis. It is difficult to differentiate cortical thrombosis from encephalitis, which in many cases may follow or be due to the former. However, it is more important to differentiate these from intracranial hypertension, which is a relatively reversible condition depending on interference with absorption of cerebral fluid through the villi rather than on venous thrombosis. This condition is revealed by edema of the ocular fundi, and the process can be arrested by proper drainage of the cerebrospinal fluid and the general elimination of fluids from the system.

Dr. Y. Muerman, Helsinki, Finland, stressed the importance of ruling out brain abscess. He has found that ventriculography will supply the desired information in questionable cases.

Dr. Francis L. Weille, Boston, recalled a case seen at the Massachusetts Eye and Ear Infirmary which was strikingly similar to one described by the authors and in which there was

general agreement among the consultants that the original focus was in the ear. Although one of the younger otologists insisted that antibiotics would cure the patient, the preponderance of opinion was in favor of surgery of the mastoid, which was carried out and was followed by recovery.

Dr. E. A. Stuart, Montreal, Canada, commenting on the points raised in the discussion, agreed that ventriculography as well as other diagnostic procedures were necessary to differentiate thrombosis from suppurative processes causing similar symptomatology. Referring to the subject of intracranial hypertension mentioned by Dr. Williams, Dr. Stuart stated that thrombosis of the superior longitudinal sinus could interfere with the drainage of the cerebrospinal fluid. He thought that Dr. O'Brien might discuss this phase.

Dr. F. H. O'Brien, Montreal, Canada, stated that the diagnosis of cerebrocortical venous thrombosis was usually arrived at by exclusion and only after the suppurative complications had been satisfactorily ruled out. As for cerebral hypertension, it was his opinion that interference with proper circulation of the cerebrospinal fluid could result as well from thrombosis of the superficial cortical veins as from thrombosis of the larger vessels, such as the superior longitudinal sinus.

Air Currents in the Upper Respiratory Tract and Their Clinical Significance. Dr. Arthur W. Proetz, St. Louis.

By means of charts, diagrams, and a motion picture film, Dr. Proetz demonstrated graphically the path of air currents, both inspiratory and expiratory, through the upper air passages. A study of these respiratory currents is of value because of their clinical significance in an evaluation of the manifestations of the common cold and other respiratory conditions. Among the many interesting facts brought out in this study are the following:

1. The soft palate and the epiglottis play important roles in directing the inspired air into its various whorls and eddies.

2. The inspired air, which is cold and dry, does not come into contact with the sinus ostia directly, whereas expiratory currents, which are warm and moist, do reach these areas. Consequently, it is likely that infections from the lower air passages can be carried to these meatuses more readily than via the inspired currents.

3. The three principle points of impingement of inspired air are in the regions of greater lymphoid tissue masses: the pharyngeal tonsil, the base of the tongue, and the faucial tonsil.

The effect of nasal abnormalities on the air currents is demonstrated, and the results of distorted currents are described. Localized hypertrophy of the mucosa and dry spots due to undue exposure to inspiratory blasts are explained and their sequelae evaluated.

Pathology, Treatment, and End-Results of Parotid Tumors. Dr. J. R. McDonald, Portland, Ore.

Histologic studies of 717 cases of parotid tumor showed 72% coming within the classification of mixed tumors. Several varieties were shown, but all possessed distinguishing characteristics, namely, epithelium, either glandular or squamous or both, in acini-like spaces, with a mesodermal mucoid connective tissue capable of differentiating into cartilage. The next most frequent tumor found was the carcinoma, of which five different types were described, and which comprised 20% of the entire series. The most malignant type was the adenocarcinoma.

Local recurrences were observed in 30% of the cases of mixed tumors, and the five-year limit is not to be relied upon as final. It is interesting to note, however, that the survival rate of these patients is almost as good as that of the average population of the same age. Only 4 patients out of 500 actually died of mixed tumor.

The cylindroma group, comprising about 5% of the series, did not fare so well. While slow growing, these tumors recur more widely than the others and continue to spread. Nevertheless, a great many of the patients survive beyond 5 and even 10 years.

As for therapy, one must be guided by the pathologist's findings. Local removal of a mixed tumor when first seen is adequate. Recurrences naturally would demand more radical surgery, even up to total parotidectomy. Malignant tumors certainly require radical surgery, with sacrifice of the nerve if necessary and with radical neck dissection.

DISCUSSION

DR. G. B. NEW, Rochester, Minn., commented on the importance of cooperation between the pathologist and the surgeon, since the surgery indicated is based on the type of neoplasm as defined by the pathologist after study of frozen sections at the time of operation. His experience so far as the mixed tumors and cylindromas were concerned coincided with that of the speaker. As for the adenocarcinomas, Dr. New found 31.5% metastasizing and the rate for the squamous cell carcinomas was still higher (53.9%). The undifferentiated carcinoma is more active, and frequently, in addition to wide excision, radium seeds may be implanted if there is any doubt as to its complete extirpation.

DR. JAMES H. MAXWELL, Ann Arbor, Mich., recalled his report of 227 cases two years previously, of which 113 were benign mixed tumors; 51 showed definite malignant change originating in a mixed tumor; 41 were primary malignant tumors. His experience was that the squamous-cell cases were the most malignant. He found that the cylindromas were most unpredictable. An analysis of 29 cases of tumors originating in the tongue, palate, nose, and salivary glands by Dr. French showed 16 patients dying of the tumor, 7 alive with recurrences or metastases, and 4 symptom-free. Pulmonary metastases are common but may remain silent for years. Dr. Maxwell favored an attempt to preserve the facial nerve in all cases. Eighty-four cases that were extensively resected showed a five-year survival rate in 68%. In 9 cases the nerve was removed, and in 13 others there was some permanent partial palsy.

Present Status of Tracheotomy in Bulbar Poliomyelitis. DR. ROBERT E. PRIEST and DR. LAWRENCE R. BOIES, Minneapolis.

Analysis of the authors' own experience plus that of a number of other observers whose publications were listed shows a preponderance of evidence in favor of tracheotomy in selected cases of bulbar poliomyelitis. In a series of 169 patients with bulbar poliomyelitis, 104 survived; 56 were tracheotomized, and of these, 25 recovered. Tracheotomy is a lifesaving measure if there is laryngeal obstruction: it is indicated to relieve atelectasis and to facilitate the removal of secretions which the patient is unable to expel. All the patients were given postural drainage, but tracheotomy was done in those instances in which the postural drainage was ineffective. It was pointed out that the resort to tracheotomy was just as feasible and as urgent as in other states in which aeration of the lungs was hampered through unconsciousness, as in head injuries, or in neurosurgical conditions, when the cough reflex is depressed, causing food or liquids to be aspirated, or when obstruction is due to paralysis or the accumulation of secretions.

Tracheotomy in Botulism and Residual Poliomyelitis. DR. HENRY B. ORTON, Newark, N. J.

Dr. Orton reported the case of a patient with botulism who was threatened with drowning in her own secretions because of her inability to expel them and who made a prompt recovery after tracheotomy. He also reported the case of a patient who recovered from poliomyelitis with a residual paresis of one vocal cord and nine months later contracted pneumonia with atelectasis and emphysema. When the respirations became labored, a mediastinotomy was done, followed by a tracheotomy. Large quantities of secretion were coughed up, and dramatic relief followed. The sudden explosive cough after tracheotomy is due to the association of vagus fibers with the sympathetic fibers.

DISCUSSION

DR. JAMES A. DOWNING, Des Moines, Ia., elaborated on the trials and difficulties he and his associates had experienced in dealing with this problem at the Raymond Blank Memorial Hospital for Children. Of 400 cases of poliomyelitis seen in 1950 and 1951, 30% were of the bulbar type. Nine tracheotomies were done in one season with only two deaths, one patient being practically at the point of death at the time of operation. Postural drainage at the 30 degree angle, suction, and administration of oxygen were carefully provided for in all cases. The difficulty of doing a tracheotomy with the patient in a respirator is well known. It has been successfully done with a bronchoscope in place delivering oxygen, and the procedure takes only five or six minutes. The rule for positive and negative pressure has been —12 mm. and

+8 mm. The latter helps clear the airway and gets the mucus out of the small bronchi. It also stops capillary bed stasis. Since the exchange of oxygen and carbon dioxide is at the beginning of expiration, when the alveoli are distended, the value of positive pressure can be appreciated when it is employed with the respirator and the expiratory rubber tube.

If a feeding tube is used, one must be careful not to force the fluids down too rapidly, because the stomach will regurgitate them, thus adding to the patient's difficulties. The drip method is the safest.

Dr. Alden Miller, Los Angeles, reviewed the experience of the contagious disease staff in Los Angeles County during the past few years. In 1948 there were 3,300 cases of poliomyelitis, and in over 200 tracheotomy was required. In 1949, tracheotomy was done in 75 of a total of 1,200 cases of poliomyelitis. Both the pediatricians and the laryngologists are convinced that the procedure has cut the mortality of bulbar poliomyelitis in half. Tracheotomy on patients in a respirator has been facilitated by the use of the Bennett positive low-sensitive pressure machine. Oxygen is given under positive pressure for inspiration and negative pressure which follows for expiration; being flow-sensitive the machine does not work when the patient breathes spontaneously. Since it is applied through a face mask, the patient can be drawn up out of the respirator, so that there is plenty of room for the surgical procedure. Last year a modification of the breathing piece was used which fits directly onto a bronchoscope, thus making the trachea more accessible for the tracheotomy.

Dr. Paul Holinger, Chicago, expressed surprise that it was necessary to start a campaign to stress the importance of a procedure as logical as tracheotomy in such conditions as those under discussion. While it is true that an intratracheal tube such as is used by anesthesiologists affords temporary relief, it must not be relied on for more than a few hours and should be considered as an excellent procedure only in an emergency and until such time as a tracheotomy can safely be carried out. Dr. Holinger has used the intratracheal tube more frequently than the bronchoscope when doing a tracheotomy on a patient in a respirator and with positive pressure from a simple bag has found it very practical. He has also used an intratracheal tube instead of the usual tracheotomy tube, which is inserted into the tracheal stoma and brought to the side of the neck where it is accessible. This is particularly an advantage in patients with short thick necks and in small infants.

Dr. Thomas C. Galloway, Evanston, Ill., commended Dr. Priest and Dr. Boies for their courage in carrying out their program against considerable initial opposition. He himself had had the same experience, and it has been a great satisfaction to find the pendulum swinging the other way as more and more observers are becoming convinced. The attempt to substitute intubation for tracheotomy at one of his hospitals had very bad results until it was given up.

It is important that the younger men be taught how to do a tracheotomy rapidly and with a minimum of dissection. It should be done high up. Dr. Galloway has never seen a complication due to a tracheotomy, not even emphysema, despite the use of the respirator.

A number of other conditions were mentioned in which tracheotomy is indicated for the relief of obstruction due to paralysis, spasm, or increase in secretions. A characteristic prototype is drowning. Such patients are frequently found with no water in the lungs, death being due to asphyxia from glottic spasm. It is only in the terminal stage of asphyxia that the glottis relaxes. Patients with tetanus, myasthenia, cerebral vascular accidents, and gunshot wounds may be carried temporarily over a crisis by intubation, but will do better if tracheotomized at the earliest moment. Tracheotomy in tetanus cases permits a lower level of curare, which is an advantage if it has to be used for more than a few days.

Dr. Robert E. Priest, Minneapolis, closed the discussion with the comment that the intratracheal tube can become occluded by inspissated secretions, thus defeating its purpose. Also, one must consider the danger of granuloma from prolonged use of the tube.

Dr. Henry B. Orton, Newark, N. J., stated that in the poliomyelitis case in which a secondary tracheotomy was required the emphysema was not due to the tracheotomy but followed rupture of the alveoli after a severe coughing spell.

Benign Tumors of the Larynx. Dr. Paul Holinger and Dr. Kenneth C. Johnston (by invitation), Chicago.

A review of 1,109 cases of benign tumors of the larynx, including specific granulomas, showed a wide variety of lesions, the commonest being the mucous polyp, which accounted for 521 of the cases. Polypoid degeneration of one or both cords was present in 112. Vocal nodules were found in 193 cases, papillomas in 115, hyperkeratotic leucoplakia in 82, nonspecific granulomas in 49, and cysts in 73. A number of colored slides were shown depicting various types of growth.

DISCUSSION

Dr. Gabriel Tucker, Philadelphia, recently went over the records of 1,196 cases of benign tumor of the larynx, with results quite similar to the findings in Dr. Holinger's series. As for stripping the vocal cords in certain cases of polypoid degeneration, Dr. Tucker would prefer a less radical procedure, even if it necessitated going back a second time. Concerning leucoplakias, it does not always follow that they should be treated as cancerous lesions. He has followed several cases for a number of years during which the lesions have remained benign despite recurrences after local excision.

Dr. Francis E. LeJeune, New Orleans, reported no success with aureomycin in treatment of papilloma of the larynx. Terramycin seems to give some promise but has not been used long enough to permit an evaluation.

Regarding a case of keratosis of the vocal cord presented by Dr. Holinger, Dr. LeJeune believes that when the lesion is very thick and white it will eventually become malignant, and he therefore would recommend wide resection followed by cauterization of the base. Dr. LeJeune prefers general anesthesia for all such operations.

Dr. Louis H. Clerf, Philadelphia, commented on the fact that a large number of the cases included in Dr. Holinger's series were not of true neoplasms. The chronic inflammatory lesions which were cited are of course very important. He has found that smoking is a very common etiologic factor. Also, in taking the history, one must be sure to inquire about exposure to other air-borne irritants which are incident to certain occupations and environments. Hyperkeratosis of the larynx is much commoner than it was years ago, and the condition is appearing in women, an occurrence unheard of before smoking became prevalent. Biopsies of keratotic areas must go deep enough to reach the basement membrane if one is to be sure that the lesion is not malignant.

Dr. Edwin N. Broyles, Baltimore, reported a case in which an extensive swollen red mass in the larynx, presenting an appearance suggestive of a carcinoma, turned out to be a cyst, probably thyroglossal. He wondered whether Dr. Holinger had encountered such a condition in his large series of cases.

Dr. Hugh G. Beatty, Columbus, Ohio, mentioned the possibility of allergy as an etiologic factor in the production of polyposis and localized edema of the cords.

Dr. W. Likely Simpson asked whether Dr. Holinger would elaborate on the apparatus he uses for the pictures he demonstrated.

Dr. Paul Holinger, Chicago, in closing, said that the pictures were shown as a part of the scientific presentation and not as a photographic demonstration, which would be a subject for some other occasion. He was amazed to note how closely his figures tallied with those cited by Dr. Tucker in his series. As for stripping the cords, he has found the procedure very necessary in keratosis, and has had to repeat it several times in certain cases before the lesions eventually became malignant. He agreed with Dr. LeJeune that the use of antibiotics is still entirely experimental. In three of the nine cases of papilloma which he reported last year the growths disappeared. Yet one cannot draw definite conclusions, because one of the patients had just reached puberty.

He cannot entirely agree with Dr. LeJeune that keratotic lesions should be excised widely, because such a procedure will not prevent the occurrence of a similar lesion in another area.

As long as the etiologic factors are operating and the soil is favorable, keratosis will recur, and, therefore, wide excision should be reserved only for cases in which malignant changes can be determined.

Concerning Dr. Broyles's case, he would be inclined to diagnose the lesion as a laryngocele because of the location; thyroglossal cysts point at the base of the tongue rather than in the larynx.

Dr. Beatty's point regarding the influence of allergy is well taken and should always be borne in mind in cases of polyposis and polypoid degeneration.

Laryngocele. Dr. Lyman G. Richards, Brookline, Mass.

Dr. Richards cited the discovery of this condition among the criers of the mosques and its later discovery among glass blowers and trumpeters. He also referred to early disagreement among observers as to pathogenesis. Anatomic studies show how the laryngocele develops from the ventricle into a sac-like protrusion resembling a Phrygian cap. The saccule is an atavistic structure in lower animals, lined with a secreting mucosa. The ventricle is more or less conical, its base directed toward the cord and its apex upward and outward. Strenuous expiratory efforts causing increased intralaryngeal pressure may force a dilatation of the saccule and the formation of a laryngocele. Three types are described: external, internal, and combined. Most frequently it appears externally just below the hyoid bone by extrusion through the thyrohyoid membrane. X-ray films assist materially in outlining the cyst. A case is reported with discussion of methods of removal, which depend on the location.

DISCUSSION

Dr. C. L. Jackson, Philadelphia, showed slides from two cases, one of which he had previously reported. Planographs showed the mass to good advantage. The external portion was removed via an external approach; the internal portion disappeared spontaneously.

Dr. H. Marshall Taylor, Jacksonville, Fla., commented on some of the characteristics of laryngocele as manifested in a patient who could produce a whistling noise by compressing the mass on the outside of his neck. The mass would then disappear but would inflate again if he swallowed a few times.

Dr. Walter B. Hoover, Boston, had seen four cases of laryngocele none of which involved persons in occupations requiring excessive blowing. In one case he was able to demonstrate a ball of thick mucus acting as a check-valve, which prevented expulsion of air from the sac after filling. Dr. Hoover objected to the classification into three types. It seems impossible to have a purely external laryngocele, since its origin must be from within. Also, he feels that all of these growths should be removed externally and that there is no need for a double operation.

Dr. Gabriel Tucker, Philadelphia, reported his experience in one case in which he did a combined internal and external operation via a laryngofissure and an external incision. He said that if he had to repeat the experience, he would prefer just the external approach.

Dr. Fred Z. Havens, Rochester, Minn., has seen small laryngoceles *in situ* in several laryngectomy specimens. He has also observed the plug of mucus mentioned by Dr. Hoover. His procedure in dealing with these lesions is to expose them externally as described, dissect the upper end free, and follow the sac down to its neck by splitting the wing of the thyroid. He is convinced that some inflammatory reaction plays a part in constricting the neck of the sac, which prevents the exit of air while not obstructing its entrance.

Dr. Lyman G. Richards, Brookline, Mass., in closing stated that Dr. Hoover's comment was as close to a disagreement as he had observed thus far in the meeting and that he was glad of an opportunity to argue about it. He was inclined to agree with him and the other speakers that one should be able to dissect out the entire sac via an external approach, even if it were lobulated into two divisions.

He conceded that inflammatory processes might cause a laryngocele, which, however, is not the same as the congenital type which may be seen even in the very young. There is still much to be learned about the true pathogenesis of the condition.

Window Laryngofissure for Carcinoma of the Larynx. Dr. Edwin N. Broyles, Baltimore.

Dr. Broyles showed a moving picture demonstrating resection of the vocal cord via a window cut through the wing of the thyroid cartilage. The procedure is indicated in isolated lesions of the cord when there is no fixation. Eighteen patients have been operated on by this method since 1938, with only one recurrence.

DISCUSSION

Dr. Louis H. Clerf, Philadelphia, inquired why Dr. Broyles prefers this method to simple thyrotomy. From the picture shown it would seem difficult to visualize the entire lesion until about the middle of the operation. Also it would appear that subglottic lesions are not so readily seen in this approach. In addition, Dr. Clerf questioned the necessity for removing the ventricular band when the cordal lesion did not reach the anterior commissure.

Dr. Paul Holinger, Chicago, expressed a preference for a laryngectomy in the type of case in which Dr. Broyles feels that his method is indicated.

Dr. Gabriel Tucker, Philadelphia, frequently does a preliminary tracheotomy and then, by introducing a scope through the opening, views the lesion from below, which affords a better opportunity of visualizing the extent of the growth.

Dr. Lyman G. Richards, Brookline, Mass., inquired how it was possible to remove the tracheotomy tube within 24 hours when there is no obvious laryngeal support at the time, and also what is the ultimate effect on the patient's voice.

Dr. Edwin N. Broyles, Baltimore, in closing, explained that his procedure affords a wider field for extensive resection. Replying to Dr. Holinger, he stated that it is barely possible that in the case shown a laryngectomy might have been considered preferable by some physicians. Certainly if a laryngectomy was done in every case of carcinoma a larger percentage of recoveries could be achieved. Still, there are many patients who recover after less radical surgery.

Dr. L. H. Clerf, Philadelphia, interrupted in order to point out that the operation is half completed before one can determine the full extent of the growth. In cases in which there is some doubt as to whether a laryngectomy is required, it would appear simpler to split the larynx before making a final decision. Also, by scooping out healthy cartilage to reach the lesion, one runs the risk of contaminating a clean area with cancer cells.

Dr. Edwin N. Broyles, Baltimore, replied that the approach is via the healthy side which affords a window through which the opposite affected cord can be viewed. Concerning the use of the retrograde laryngoscope suggested by Dr. Tucker, he believed it to be a fine idea, but unfortunately he had lost his laryngoscope while in military service and had used none since. Another advantage of his approach through the healthy thyroid wing is that damage to the anterior commissure tendon is thus avoided.

Concerning Dr. Richards' question about breathing space, he has never encountered any difficulty, since it is apparent that the strap muscles, the hyoid above and the cricoid below, maintain an adequate airway despite loss of one thyroid ala. Patients do experience some difficulty in swallowing for the first day, but they can prevent aspiration of fluids by turning the head to the good side.

Modern Trends in Otolaryngology with Special Emphasis on Allergy. Dr. Rea E. Ashley, San Francisco.

Dr. Ashley discussed allergy as a phenomenon of autonomic imbalance resulting from various types of stimuli—protein, and emotional, and traumatic—on a system in which the threshold of tolerance is lower than average. Increased permeability of the blood vessels and cells leads to loss of fluid and electrolytes. A variety of symptoms referable to the ear, nose, and throat results. Nasal obstruction, postnasal discharge, chronic cough, and symptoms suggesting sinus infection are very common. Diagnosis rests on the personal and family history, together with the presence of large numbers of eosinophiles in the secretions and the tissues,

and frequently specific sensitivity to various types of allergens. The common cold may be allergic in its earliest stages, and frequently is ameliorated by antiallergic therapy. There is a possibility that corticotropin (ACTH) and cortisone may influence the organs responsible for oversensitivity. Good results were obtained in six cases of asthma. Hormone stimulation of the sympathetic nervous system may restore the balance between it and the parasympathetic system.

DISCUSSION

Dr. Henry L. Williams, agreed that the subject is of prime importance, although as yet not clearly understood. Granting that advances have been made in the study of the physiologic mechanism of diseases of the ear, nose, and throat, there is still a great deal that has to be learned concerning the alterations and dysfunctions of the peripheral vascular bed, interstitial collections of fluid, electrolyte metabolism, endocrines, and the lymph flow, all of which seem to be factors in the general picture of allergy. Much of present-day therapy, while effective in some cases, is illogical in others because of the incompleteness of our knowledge This is clearly demonstrated by the conflicting reports on the use of antihistamines in treatment of the common cold and the hit-or-miss attempts to employ pituitary and adrenal hormones in our specialty. As the basic problems under study become more clearly understood, further advances in therapy can be expected to yield better results.

Dr. Aubrey G. Rawlins, San Francisco, stressed the importance of calculating the absolute number of circulating eosinophiles when one is giving corticotropin or cortisone. As an example, he cited a case of asthma in which the eosinophile count was 700, considered not excessively high. After five days of corticotropin therapy in fairly good-sized doses the asthma continued and the count was unchanged. This indicated that the adrenal cortex was not working properly. Corticotropin stimulated the adrenal cortex but failed in this case, since no cortisone was released into the circulation. With a change in therapy to administration of cortisone the symptoms disappeared within 24 hours and the eosinophile count was reduced.

Another point was raised which is somewhat theoretical and has to do with the mechanism of low doses in desensitization: the fact that although patients improved and were relieved, subsequent skin testing revealed a reaction as definite in size as the original. This would seem to indicate that the action of the therapy is not through the building up of blocking antibodies but probably through a sort of alarm reaction and that the injections evidently stimulate the production of cortisone.

Dr. F. L. Weille, Boston, inquired whether Dr. Ashley was familiar with a recent theory relating to the probable etiologic relationship of peptone to allergic reactions in contrast to histamine; also, what explanation can be given for persisting allergic symptoms despite elimination and desensitization therapy. He was interested in what Dr. Ashley does for patients with vasomotor rhinitis when there is no demonstrable allergy, but when all the usual symptoms including eosinophiles are present.

Dr. Rea E. Ashley, San Francisco, in closing stated that he had had no acquaintance with the peptone theory, but hoped to find out about it. Regarding the vasomotor cases mentioned by Dr. Weille, he makes frequent examinations for eosinophiles and pus cells and tests for house dust, tobacco smoke, newspaper, molds, etc.; and even if the tests are negative, he gives injections on an empirical basis and, in addition, cauterizes the turbinate bones. The results in a large percentage of cases justify the procedure.

Hypertrophy of the Masseter Muscle: A Clinical Entity. Dr. James H. Maxwell, Ann Arbor, Mich.

Dr. Maxwell reported four cases of hypertrophy of the masseter muscle, mostly unilateral; three of the patients were young females. The subjective symptoms were fatigue on chewing, with some local soreness. Objectively they presented an asymmetry of the jaw which could be seen in roentgenograms and might have been mistaken for a parotid tumor. Electromyograms were not conclusive, and biopsies of the muscle showed some changes in the fibers with diminution in the number of striations, although this was not a constant finding. As to etiology, it is possible that the hypertrophy results from the habit of grinding the teeth.

DISCUSSION

DR. NORTON CANFIELD, New Haven, Conn., asked whether the hypertrophy involves the entire muscle or just the portion at the angle of the jaw.

DR. HARRY P. SCHENCK, Philadelphia, inquired whether biopsy specimens were taken from the opposite masseter.

DR. G. B. NEW, Rochester, Minn., commented on the fact that the roentgenograms in two cases showed unerupted third molars and he wondered whether that finding had any significance.

DR. JAMES M. ROBB, Detroit, asked whether the patients were all young women.

DR. JAMES H. MAXWELL, San Francisco, in closing, stated that apparently the entire muscle was affected. It was remarkable in these cases to note that when the patient clenched the jaw the whole muscle bulged outward into a fully palpable mass. It was definitely not a hernia through the sheath. Regarding biopsy of the opposite masseter, it is difficult to express an opinion, because pathologists cannot agree as to what the normal number of striations should be. It has been stated that a large number of cases would have to be observed and the results tabulated before one could make a positive statement on that point.

Regarding the role of unerupted third molars, it was noted in three persons that the swelling began two or three months after the extraction of an unerupted molar.

Mikulicz' disease and sarcoidosis were both excluded in Dr. Maxwell's cases.

General neurological examination also failed to determine the presence of a tendency toward muscular dystrophy. In reply to Dr. Robb's question, Dr. Maxwell stated that the patients were all young women except one, an 11-year-old boy. Their ages were below 30.

Sarcoidosis: Ear, Nose, and Throat Complications. DR. JOHN R. LINDSAY and DR. HENRY B. PERLMAN (by invitation), of Chicago.

Nine cases of sarcoidosis in which lesions appeared in the nose and throat were thoroughly studied and documented. Biopsies of the inferior turbinate in eight of the cases revealed typical sarcoid findings. Some of the lesions disappeared spontaneously, while others persisted unchanged for as long as five years. Kodachrome® slides of these lesions were shown, depicting the histologic changes observed. The nasal mucosa presented pale miliary tubercles in a hypertrophic mucosa with crusting and discharge. Occasionally larger granulomas were found.

A thorough résumé of the history of the disease was presented, pointing out its differentiation from lupus and tuberculosis. The lesions are widely disseminated, involving the lungs, lymph nodes, eyes, skin, and bones, and the progress of the disease is chronic with usually very little constitutional disturbance. The intracutaneous tuberculin test gives a negative reaction as a rule, and the gamma globulins are often increased. The lesions consist of epithelioid tubercles with little or no necrosis and with refractile or calcified bodies in the giant cells.

One patient treated with vitamin D failed to respond.

DISCUSSION

DR. SUBRAMIAN, Madras, India, reported having seen an interesting case recently in which a man of 30 complained of a dry cough and a choking sensation on swallowing. Examination revealed a mass at the base of the tongue which looked like a carcinoma, but on biopsy was reported to show tubercles, giant cells, etc. Chest roentgenograms revealed an apparent miliary tuberculosis which, however, was not substantiated by the bacteriologic examination. In a conference among laryngologist, chest specialist, and pathologist, it was concluded that it was a case of sarcoidosis of the lingual tonsils and lungs.

DR. J. R. MAXWELL, San Francisco, stated that he had seen two cases of sarcoidosis during the past 18 months, one with bilateral parotid involvement and the other with predominantly laryngeal. Diagnosis was confirmed by histologic examination of biopsy specimens. The lesions disappeared in four months during treatment with large doses of vitamin D. Relative to the case reported by Drs. Lindsay and Perlman, in which a patient given a diagnosis of having had sarcoidosis later died of tuberculous meningitis, he offered his experience with a patient who had an enlarged submaxillary gland removed which was reported as showing sarcoidosis, but who some years previously had had some cervical nodes removed which were definitely tuberculous.

With this information at hand, the pathologist made more sections of the submaxillary gland and finally concluded that it might have been tuberculous.

Dr. T. C. Galloway, said he had obtained satisfactory results from the use of roentgen rays and inquired whether Dr. Lindsay and Dr. Perlman had employed this therapy in their cases.

Dr. R. E. Priest, Minneapolis, referring to the use of large doses of vitamin D, called attention to a case of lupus vulgaris of the nasal septum, which he reported a year or so ago, in which the patient was treated with very large doses of synthetic vitamin D, called calciferol. He advised that care be taken in the use of this agent, because it is potentially dangerous to the kidneys through the laying down of calcium.

Dr. N. Canfield, New Haven, Conn., recalled a case in which the lung lesions appeared and disappeared at least six times. The patient, who also had lesions in the nasopharynx, hypopharynx, and larynx, was followed for 15 years with various ups and downs. Dr. Canfield inquired whether the authors had had any experience with the use of urethan in these cases.

Dr. R. C. McNaught, San Francisco, had a case in which a swelling of the bridge of the nose following a submucous resection was reported as sarcoidosis. Cortisone was given with apparently very successful results.

Dr. Henry B. Perlman, Chicago, in closing, replied to Dr. Subramian that when the lesions become very large they may necessitate surgery. The vitamin D therapy seemed to improve the superficial lesions but failed to influence the deeper lesions. In the case of the patient who died of tuberculous meningitis, the tuberculin test was negative until the terminal stage. He had a positive Kveim reaction, and it was the combined opinion of the chest specialists and the pathologist that is was possible for the patient to have both sarcoidosis and tuberculosis. Dr. Perlman has had no experience with roentgen therapy in these cases. Concerning the use of calciferol, he had employed it in one case of lupus vulgaris without seeing any improvement, although the skin lesions did recede. He had had no experience with cortisone although he is aware of its use by men in the chest department.

News and Comment

AWARDS FOR ESSAYS ON PLASTIC AND RECONSTRUCTIVE SURGERY

The Foundation of the American Society of Plastic and Reconstructive Surgery announced the winners of the 1951 awards in its competitive essay at the Annual Meeting of the Society in Colorado Springs, Oct. 30 to Nov. 1, 1951. Dr. Richard B. Stak, New York City, won a scholarship award for his essay, "The Cross-Leg Flap Procedure." Dr. Kathryn Lyle Stephenson, Santa Barbara, Calif.; Dr. Michael N. Tempest, Leeds, Yorkshire, England, and Dr. Allyn J. McDowell, North Hollywood, Calif., received honorable mention for their essays on "The Production of Ectopic Cartilage," "Cross-Finger Flaps in the Treatment of Injuries to the Finger Tip," and "Mass Treatment of Burns in Atomic Warfare," respectively. Information concerning the 1952 Competitive Essay may be secured from The Awards Committee, % Dr. Jacques W. Maliniac, 11 East 68th Street, New York 21.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The next meeting of the Central Illinois Society of Ophthalmology and Otolaryngology will be held at the Abraham Lincoln Hotel in Springfield, Ill., on March 28, 29, and 30. The guest speakers will be Dr. James B. Costen of St. Louis and Dr. Trygve Gundersen of Boston.

ANNOUNCEMENT OF EXAMINATIONS

It has been announced by the American Board of Otolaryngology that it will conduct the following examinations in 1952:

May 13 to 17, in Toronto, Canada, at the Royal York Hotel;

Oct. 6 to 10, in Chicago, at the Palmer House.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

As of Feb. 1, 1952

Audicon Models 400 and 415

Mfr., National Earphone Co., Inc.
20 Shipman St.
Newark 2, N. J.

Audivox Model Super 67

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
259 W. 14th St.
New York 11

Aurex Model F

Aurex Model H

Aurex Models L and M

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Harmony Mono-Pac

Beltone Symphonette Model

Beltone Mono-Pac Model M

Mfr., Beltone Hearing Aid Company
1450 W. 19th St.
Chicago 8

Cleartone Model 500

Cleartone Model 700

Cleartone Regency Model

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Mfr., The Dahlberg Company
2730 W. Lake St.
Minneapolis 16

Dysonic Model 1

Mfr., Dynamic Hearing Aids, Inc.
1042 Atlantic Ave.
Brooklyn 16

Electroear Model C

Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Model V-35

Gem Model V-60

Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Maico Atomeer

Maico UE Atomeer

Maico Quiet Ear Models G and H

Maico Model J

Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal and Magnetic) Aurophone Model 200

1947-Mears Aurophone Model 98

Mfr., Mears Radio Hearing Device Corp.
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Star Model

Mfr., Micronic Company
123 Worcester St.
Boston 18

Microtone Audiomatic T-5

Microtone Classic Model T-9

Microtone Model T-10

Microtone Model T612

Microtone Model 45

Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506

Mfr., National Hearing Aid Laboratories
815 S. Hill St.
Los Angeles 14

Otarion Model E-4

Otarion Models F-1, F-2, and F-3

Otarion Model G-2

Mfr., Otarion, Inc.
159 N. Dearborn St.
Chicago 1

Paravox Model J (Tiny-Myte)

Paravox Model XT ('Xtra-Thin)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC, and YC-7) (Veri-Small)

Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Permo-Magnetic (Multipower)

Radioear Permo-Magnetic (Uniphone)

Radioear All-Magnetic Model 55

Radioear Model 62 Starlet

Radioear Model 72

Mfr., E. A. Myers & Sons
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Distr., Radioear Corporation

Rochester Model R-1

Rochester Model R-2

Mfr., Rochester Acoustical Laboratories, Inc.
117 4th St., S. W.
Rochester, Minn.

Silvertone Model 103BM

Mfr., National Hearing Aid Laboratories
815 S. Hill St.
Los Angeles 14

Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Silvertone Model J-92

Mfr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Silvertone Model P-15

Mfr., W. E. Johnston Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99

Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Model 700

Sonotone Model 900

Sonotone Models 910 and 920

Sonotone Model 925

Sonotone Model 940

Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E

Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Model 22

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 1700

Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal

Tonemaster Cameo Model

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Trimm Vacuum Tube Model 300

Mfr., Trimm, Inc.
400 Lake St.
(P. O. Box 489)
Libertyville, Ill.

Unex Model A

Unex Midget Model 95

Unex Midget Model 110

Unex Models 200 and 230

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Model J

Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 and 66

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
259 W. 14th St.
New York 11

Zenith Model 75

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex (Semi-Portable)

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid

Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50

Mfr., Sonotone Corporation
Elmsford, N. Y.

Directory of Otolaryngological Societies *

INTERNATIONAL

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

Meeting: Third Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology
Place: Habana, Cuba. Time: January 20-24, 1952.
President: Dr. José Gros.
Secretary: Dr. Pedro Hermandez Gonzalo, Calle 8, 358, Habana, Cuba.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

Place: Caracas, Venezuela. Time: 1953.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: Chicago. Time: June 11-13, 1952.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 14-20, 1951.

AMERICAN BOARD OF OTOLARYNGOLOGY

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 6-10, 1952.
Place: Royal York Hotel, Toronto, Canada. Time: May 13-17, 1952.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Royal York Hotel, Toronto, Canada. Time: May 20-22, 1952.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 2.
Place: Royal York Hotel, Toronto, Canada. Time: May 23-24, 1952.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Royal York Hotel, Toronto, Canada. Time: May 20-22, 1952.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Place: Waldorf-Astoria, New York. Time: Jan. 11, 1952.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Place: Academy of Medicine, Atlanta. Time: Jan. 14, 1952.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Place: Indianapolis Athletic Club, Indianapolis. Time: Jan. 28, 1952.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.
Place: County Medical Building, Los Angeles. Time: Jan. 19-20, 1952.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Royal York Hotel, Toronto, Canada. Time: May 18-19, 1952.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. William H. Evans, 24 Wick Ave., Youngstown 3, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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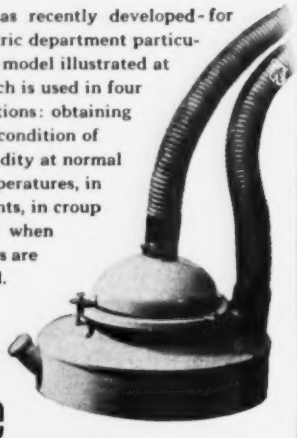

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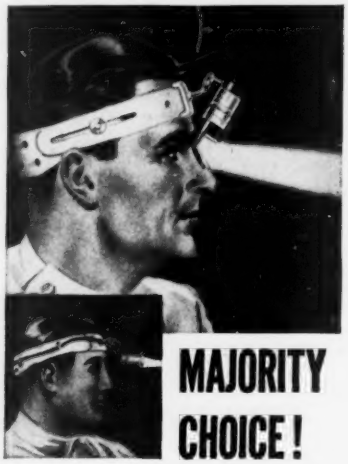

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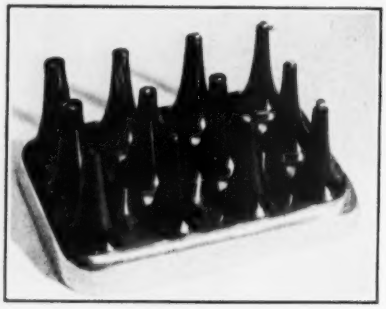

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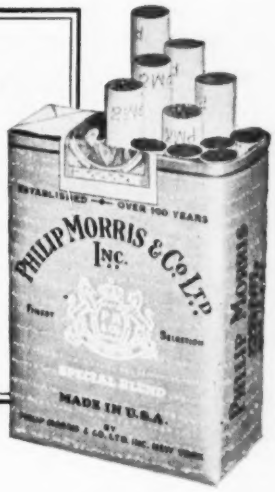

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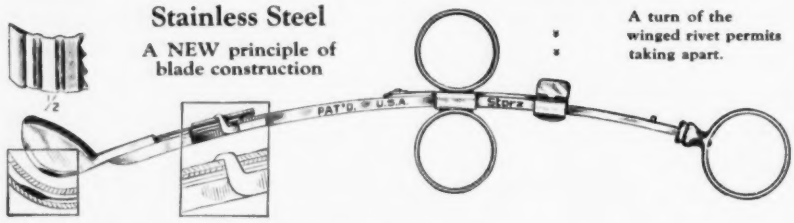

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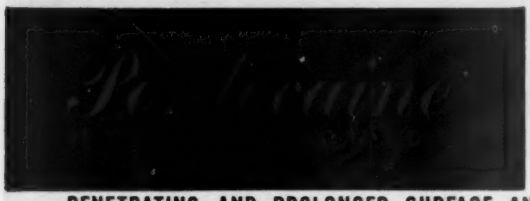

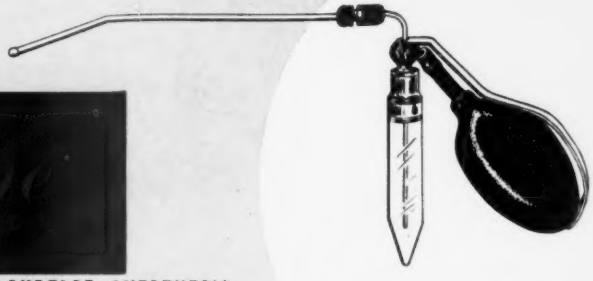

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1. Carabelli, A.: *Diseases of the Chest*, 15:532, May, 1949.

2. George P. Pilling & Sons, Company, 3451 Walnut Street, Philadelphia 4, Pa.

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